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SALICYLAMIDE
(BENGER)

Recent intensive research indicates that in Salicylamide Benger (ortho-hydroxy benzamide) the clinician has a powerful weapon against pain—one that permits the massive doses not possible with other analgesics.

ANALGESIC-ANTIPYRETIC ACTION

Hart. E. R. (1946) Bull. Fed. Ass. Soc. Exp. Bio. 5, 182, has shown that Salicylamide possesses an analgesic potency 7½ times that of aspirin. This agrees with the clinical findings of Litter *et al.* (1951) J. Pharmacol, 101, 119 who treated 40 cases of rheumatoid arthritis, 17 of osteo-arthritis, 54 of fibrositis and 7 of rheumatic fever, and in half this group, the analgesic effect was described as "marked".

Salicylamide (Benger) is at least as effective as aspirin in reducing febrile conditions and its low toxicity enables the clinician to employ high therapeutic doses.

ABSENCE OF TOXICITY

Numerous workers have demonstrated that the drug is well tolerated and in contra-distinction to salicylate therapy, prolonged and massive doses do not produce—

(1) gastric irritation
(2) renal damage
(3) changes in prothrombin times, or in erythrocyte and haemoglobin values.

REFERENCES:

(1) Seeberg, V.B. *et al* (1951) J. Pharmacol, 101, 275.
(2) Hofman H., Neubauer M. Deutsche Gesundheitwesen 5:776 June 1950.
(3) Euler. E., Remy R., Med. Klin. 45(37): 1,178, 1950.

APPLICATION

Salicylamide (Benger) has no unpleasant taste and permits the use of high doses without undesirable side-effects It may be used as a powerful general analgesic and in the treatment of rheumatic fever and other degenerative and inflammatory diseases of joints, muscles and ligaments. Reports of its use in various neuralgias are encouraging.

DOSAGE. The dose should be adjusted according to the patient's response (8–12 gms. per day have been used over prolonged periods without undesirable side-effects) The average effective adult dose is 2 gm. every four hours, night and day.

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DIE HEDENDAAGSE BEHANDELING VAN KONGENITALE MEGAKOLON

(DIE SIEKTE VAN HIRSCHSPRUNG)

F. D. DU T. VAN ZYL, CH.M., F.R.C.S.
Kaapstad

Sedert baie jare reeds word die siekte van Hirschsprung (kongenitale megakolon) toegeskryf aan 'n gebrek aan koordinasie tussen die sympathiese en parasympathiese dele van die autonomiese senustelsel; dit is dus beskou as 'n vorm van ‚akalasie'. Dié beskouing word gesterk deur die waarneming dat in hierdie pasiënte die blaas dikwels ook vergroot is en tekens van abnormale funksie vertoon. Die ontledigingsaksie van beide die blaas en rektum word deur die sakrale gedeelte van die parasympathiese senustelsel beheer.

Swenson het egter onlangs vasgestel dat die gebrek aan koördinasie nie bloot funksioneel is nie, maar dat dit die gevolg is van 'n organiese abnormaliteit wat daar bestaan. Hy het gevind dat daar 'n gebrekkige ontwikkeling van die plexus van Auerbach in die wande van beide rektum en distale gedeelte van die sigmoïede kolon is. Hierdie gebrek strek sig soms nog hoër langs die kolon uit. Die parasympathiese beheer oor die dermkanaal word deur middel van die plexus van Auerbach uitgeoefen. Stimuli word na die grootste gedeelte van die kanaal tot by die kolon descendens langs die vagus senuwees aangevoer, maar laer af kom hulle langs die pelviese nervi, nl. die nervi erigentes.

Tot dusver is dit nog nie vasgestel nie of die organiese parasympathiese gebrek alleenlik in die plexus van Auerbach voorkom, en of dit moontlik hoër op, nl. in die sakrale gedeelte van die rugmurg gesoek moet word, waar die sentrale senuselle lê.

Die feit dat beide die blaas en die rektum so dikwels in dieselfde pasiënt aangetas is, laat 'n mens 'n meer sentraal geleë letsel vermoed.

In die lig van hierdie bevindings is dit sterk te betwyfel of ons nog in hierdie gevalle die bestaan van 'n sigmoïedo-rektale sphincteriese aksie kan aanvaar. Die geloof aan die bestaan van so 'n sphincter spruit juis uit die makroskopiese beeld van vernouing in die sigmoïedo-rektale gedeelte van die derm, eerder dan uit 'n werklike demonstrasie van die bestaan van so 'n sphincter.

Die vernouing in gevalle van kongenitale megakolon strek oor 'n lang afstand en kan die hele rektum, die sigmoïede kolon en selfs die kolon descendens insluit. In tipiese gevalle egter sluit dit net die sigmoïedo-rektale gebied in.

Die proximale gedeelte van die kolon is as 'n sekondêre gevolg verwyd en die spier van die dermwand is gehipertrofeer. Die abnormale voorkoms wat aanleiding gee tot die enigsins misleidende naam, megakolon, is geensins toe te skryf aan 'n ileus nie. Die uitgesette deel van die derm vertoon normale aktiewe peristalse en dit is vergroot bloot as gevolg van die gedeeltelike obstruksie, funksioneel van aard, in die distale vernoude segmente, waar, by deurligting die gebrekkige peristalse duidelik waarneembaar is.

Indien hierdie waarnemings dus korrek is behoort die verwydering van die kronies, obstruktiewe gedeelte aanleiding te gee tot 'n inkrimping van die vergrote kolon. Waar 'n kolostomie in sulke gevalle gedoen is, het die proximale gedeelte homself dan ook spoedig ontlas en ingekrimp tot normale omvang.

Dieselfde resultaat word verkry deur reseksie van die vernoude distale segmente, indien genoegsaam verwyder word om te verseker dat daar nie derm oorbly met gebrekkige parasympathiese toevoer nie. Waar 'n onvoldoende reseksie gedoen word kan 'n herhaling van die simptome stellig verwag word. In die praktyk is gevind dat die reseksie tot minstens twaalf centimeters bokant die hoogste grens van die vernouing moet strek. Daar is tans kinders wat, na sulke reseksies, reeds 3 jaar lank van enige simptome van obstruksie ontslae is. Die goeie resultate is verder só konstant dat die wisselvallige resultate van ander operatiewe behandelings, wat vroeër toegepas is, soos bv. lumbale ganglionektomie en totale kolektomie, glad nie daarmee vergelyk kan word nie.

Die operatiewe mortaliteit is gering, veral as mens in aanmerking neem dat die pasiënte gewoonlik minder as 2 jaar oud is en boonop nog dikwels tingerig is as gevolg van die herhaalde aanvalle van obstruksie.

Hierdie lae mortaliteit word veral bevorder deur die versigtige en deeglike voorbereiding vir die operasie deur middel van dermspoelings, die toediening van antibiotika en die korreksie van enige bloedarmoede en elektrolitiese afwykinge. Verder is die tegniek van die operasie ook sulks dat slegs die mobilisasie van die gedeelte wat verwyder moet word, in die buikholte geskied, terwyl die werklike amputasie en herstel van die dermkanaal buitekant die buik, naamlik op die perineum, geskied. Op dié wyse kan 'n reseksie tot vlak bo die sphincter van die anus gedoen word met 'n minimum van trauma en besmetting van die buikholte.

Hierdie tegniek is eerste deur Swenson [1] beskryf en is 'n wysiging van die sogenoemde ‚deurtrek'-operasie vir

reseksie van die rektum met behoud van die sphincters van die anus. Hiatt [2] het hierop verbeter deur eers 'n kunsmatige intussussepsie te bewerkstellig en dan die ingewand af te sny en weer te las op dieselfde wyse as wat gedoen word in die operatiewe behandeling van 'n prolapsus van die rektum. Daar word gelyktydig in die buik en op die perineum gewerk. Terwyl die chirurg die amputasie en anastomose by die anus doen, herstel sy assistent die peritoneale bedekking van die vloer van die bekken en sluit die buik. Op dié wyse word die tydsduur van die operasie aansienlik verkort.

Die baba was by ondersoek normaal in alle opsigte behalwe vir die vergrote buik. Peristalse kon duidelik gesien en gevoel word. By rektale ondersoek is gevind dat die sphincters van die anus ietwat gespanne was.

Radiologiese ondersoek by 'n tweede geleentheid, toe 'n versigtiger tegniek toegepas is, het aangetoon dat daar 'n lang onreëlmatige spastieke en enge rektum en rekto-sigmoïed is. Daarbo, egter, word die kolon op tregtervormige wyse, tipies van hierdie siekte, wyer. Die

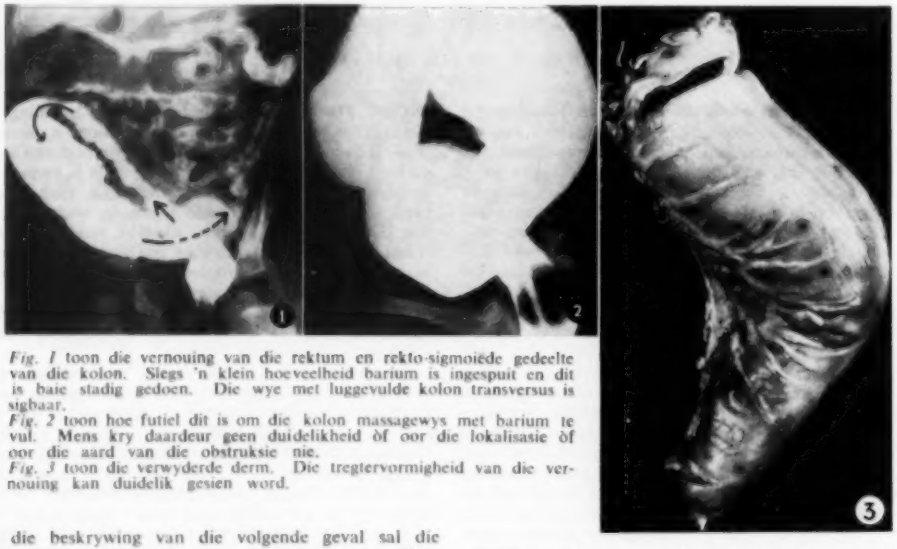

Fig. 1 toon die vernouing van die rektum en rekto-sigmoïede gedeelte van die kolon. Slegs 'n klein hoeveelheid barium is ingespuit en dit is baie stadig gedoen. Die wye met luggevulde kolon transversus is sigbaar.
Fig. 2 toon hoe futiel dit is om die kolon massagewys met barium te vul. Mens kry daardeur geen duidelikheid òf oor die lokalisasie òf oor die aard van die obstruksie nie.
Fig. 3 toon die verwyderde derm. Die tregtervormigheid van die vernouing kan duidelik gesien word.

Uit die beskrywing van die volgende geval sal die besonderhede o.a. ook die van die tegniek van Hiatt meer duidelik blyk.

Die pasiënt is 'n blanke seuntjie van 9 maande. Kort na 'n normale geboorte is bemerk dat die baba se buik opgesit was en na 3 dae het hy nog geen mekonium passeer nie. Na radiologiese ondersoek is vasgestel dat die distale kolon geweldig vergroot is. Die barium is egter in massa-vorm ingelaat en die vergrote kolon vol barium het die buik so gevul dat geen vernoude gedeelte onderskei kon word nie.

Na veel gesukkel het 'n volledige ontlasting gevolg en vir etlike maande het dit redelik goed gegaan. Daarna het hy toe meer en meer hardlywig geword en twee keer het daar 'n tydelike akute obstruksie ontwikkel. By elkeen van dié geleenthede het die buikie letterlik soos 'n tamboer gespan en 'n noodoperasie vir verligting is by beide geleenthede ernstig oorweeg. Na die jongste aanval is besluit om oor te gaan tot 'n radikale operasie alhoewel die kind toe nog maar 9 maande oud was.

'n Voorlopige kolostomie is veral vermy daar dit reeds gevind is dat die vernouing van die kolon wat volg op so 'n kolostomie tot 'n enge anastomose lei wat tot 'n werklike stenose mag oorgaan. 'n Kolostomie is werklik ook onnodig onder moderne omstandighede, te meer waar die tegniek van Hiatt toegepas word.

proximale gedeelte van die sigmoïede kolon word dan 'n ware mega-sigmoïed, terwyl die kolon hoër op vol gas is en ewe dik vertoon. By die tegniek wat hier toegepas is laat mens die barium baie stadig in die rektum vloei, sodat daar nooit 'n groot versameling van kontrasmedium is wat die hele gesigsveld kan uitwis nie.

Voorbereiding vir Operasie. 'n Geringe mate van bloedarmoede is deur 'n bloedoorgieting gekorrigeer.

As verdere voorbereiding het die baba reeds oor 'n geruime tyd aanvullende vitamine preparate gekry en vir 5 dae voor die operasie het hy ekstra glukose en aminosure in die vorm van Procasinol (een teelepelvol *t. i. d.*) gekry. Terselfdertyd is ook die volgende middele per mond toegedien met die doel om die kolon te steriliseer en aldus na-operatiewe verwikkelinge te voorkom.

1. Penicillien tablette: 100,000 eenhede elke 4 uur.
2. Streptomysien: 0.1 gm. elke 4 uur.
3. Terramysien: 0.1 gm. elke 4 uur.
4. Sulphamethazien: 1 teelepelvol drie maal daagliks.

Vir die 5 dae voor die operasie is boonop ook volgehou met die daaglikse kliesma van een pint melk waarby een ons glycerine gevoeg is. Hierdie behandeling is reeds na die jongste aanval van akute obstruksie begin.

Operasie. Die narkose, deur dr. E. G. van Hoogstraten toegedien, het bestaan uit ether, gas en suurstof met 'n byvoegsel van 60 mg. Flaxedil.

Die eerste stap was om 'n sagte rubber kateter in die blaas te sit. Die kateter is nie weer verwyder voor die negende dag na die operasie nie. Rede hiervoor is om urine-retensie te voorkom.

Daarna is 'n intraveneuse kanulle in die regterkantse vena saphena major geplaas vir die toediening van vloeistof en heel eerste is Dextran teen 'n spoed van 8 druppels per minuut gegee. Later gedurende die operasie is die Dextran vervang deur bloed, waarvan 'n totale hoeveelheid van 400 c.c. gedurende en na die operasie gegee is teen 'n snelheid van tussen 6 en 8 druppels per minuut.

Op dié stadium is die pasiëntjie in 'n semi-lithotomiese posisie geplaas en beide die abdominale en perineale operasievelde voorberei, maar op so 'n wyse afgedek dat daar 'n behoorlike skeiding tussen die twee was.

Die buik is toe deur 'n lae paramediane incisie aan die linkerkant geopen, en hierdeur het die geweldige sigmoïede kolon onmiddellik tevoorskyn gekom. Nadat dit opgelig is, het die tregtervormige vernouing van die sigmoïedo-rektale gedeelte duidelik sigbaar geword.

Die res van die ingewande is toe afgesonder en in die boonste gedeelte van die buik geplaas. Die sigmoïed is toe na links getrek en die bloedvate in die mesenterium kon duidelik herken word, omdat daar slegs weinig vet in die mesosigmoïed van sulke klein kinders is.

Die arteria haemorrhoidalis superior en die verskeie arteria sigmoïdea is toe uitgeken en tesame met hul vene digby hul oorsprong uit die arteria mesenterica inferior, dubbel afgebind en deurgesny. Op dié wyse is 'n aantal van die vaatboë in die mesenterium behou en sodoende is 'n voldoende bloedtoevoer verseker na die gemobiliseerde sigmoïede kolon, wat eindelik 12 cm. bokant die vernoude gedeelte afgesny word.

In die volgende stadium is die peritoneum van die bekkenholte deurgesny en losgemaak en 'n begin gemaak met die mobilisasie van die rektum, eers aan die agterkant en daarna aan die voorkant. Hierdie disseksie word met die grootste omsigtigheid so na as moontlik aan die muskulêre wand van die rektum gedoen, met die doel om die omliggende parasympathiese senuwees, waarvan die funksie van die blaas afhanklik is, so min moontlik te beseer, en aldus die moontlike ontwikkeling van 'n urine-retensie na die operasie te probeer voorkom. In die loop van die disseksie is die laterale ligamente van die rektum deurgesny en die twee middelste arteria haemorrhoidalis afgebind en deurgesny, en die disseksie deurgevoer tot op die levatores ani. Gedurende die hele proses was daar geen noemenswaardige bloeding nie.

Volgens die metode van Hiatt (Swenson sou op hierdie stadium die rektum deursny) steek die tweede assistent nou 'n Allis' weefseltang deur die anus in die rektum op, onder versigtige begeleiding van sy voorvinger in die rektum. Met behulp van die chirurg word die instrument nou hoër opgestoot tot op 'n voorafbepaalde punt in die sigmoïed. Hier word die wand van die derm nou van binne vasgeknyp en word die tang weer na benede getrek terwyl die chirurg van bo af help om die derm te laat teleskopeer. Na 'n bietjie gesukkel is op dié wyse 'n volslae intussussepsie van die sigmoïed en die rektum bewerkstellig sodat hierdie gedeeltes van die dermkanaal eindelik by die anus uitgehang het.

Die chirurg het toe oorgestap na die perineale operatiewe veld terwyl die eerste assistent die peritoneale bedekking van die bekken herstel het, nadat hy 'n rubber dreineringsbuis in die retrorektale holte geplaas het. Die buikwand is daarna om die buis gesluit. Hiatt verkies om die dreineringsbuis na onder te lei deur 'n aparte incisie tussen die coccyx en die anus.

Nadat die uithangende derm, wat nou natuurlik met sy slymvlies na buite gekeer was, deeglik ontsmet is, is 'n begin gemaak met die amputasie daarvan. 'n Kort snytjie is eerstens deur die intussussipiens (bestaande uit rektum) en intussusseptum (bestaande uit sigmoïed) gemaak op 'n afstand van 2.5 cm. onderkant die anus. Hier is die intussusseptum en intussussipiens toe aanmekaar geheg—peritoneum aan peritoneum en slymvlies aan slymvlies. 'n Ooglose naald met 00 katgut is gebruik en die anastomose was deurgaans deur middel van onderbroke hegtinge op 'n afstand van ¼ duim van mekaar geplaas. Die incisie is toe trapsgewyse rondom die prolapsus verleng en na elke verlenging is die nuwe stukkie eers weer geheg. Hiermee is volgehou totdat die hele omvang voltooi was.

Na dit klaar was het daar 'n stompie rektum 2.5 cm. lank met sigmoïede kolon daaraan geheg, by die anus uitgesteek. Die anastomose is toe in die liggaam teruggedruk en die operasie is voltooi deur die inplasing van 'n rubber kateter om flatus uit te laat.

Die tydsduur van die operasie was ongeveer anderhalf uur.

In die na-operatiewe tydperk is volgehou met die bloedtransfusie totdat 'n totaal van 400 c.c. toegedien was, waarna daar oorgeslaan is na soutoplossing met glukose. Sodra die baba behoefte daarvoor getoon het is klein hoeveelhede water per mond gegee, en na 24 uur is verdunde melkvoedings gegee.

Verder is 10,000 eenhede Penicillien en 0.1 gm. Streptomysien elke 6 uur per inspuiting toegedien en binne 24 uur is weer begin met die toediening van die sulphamethazien per mond soos in die voor-operatiewe dae. Teen die vierde dag kon die gewone vol-sterkte melkvoedings reeds weer gegee word.

Deur die kateter wat in die nuwe ‚rektum' gelaat is, is op die vierde dag 'n klein hoeveelheid glycerine gespuit, en daarop het toe 'n klein stoelgang gevolg.

Op die vyfde dag is die abdominale dreineringsbuis uitgetrek en vanaf daardie dag was daar daagliks van een tot drie normale stoelgange.

Teen die negende oggend het die pasiëntjie reeds vanself teen die traliewerk van die bedjie opgesukkel en op die dertiende dag kon hy uit die hospitaal ontslaan word.

Opsomming. 'n Beskrywing word gegee van die hedendaagse idees oor die pathologie van die siekte van Hirschsprung. Die operatiewe behandeling word beskryf en 'n tipiese geval word gerapporteer.

Summary. The pathology of Hirschsprung's disease is described and the present-day operative treatment indicated. A typical case is reported.

VERWYSINGE

1. Swenson (1950): Surgery, **28**, 371.
2. Hiatt (1951): Ann. Surg., **133**, 321.

NEW PREPARATIONS AND APPLIANCES

MENSTROGEN TABLETS

Menstrogen Tablets are a combination of Ethinyl Oestradiol 0.01 mg. and Ethisterone (orally effective Progesterone) 10 mg.

They are intended primarily for the treatment of secondary amenorrhoea, the rationale being based on Zondek's discovery that a correctly proportioned quantity of oestrogen and androgen administered simultaneously would not only reduce the requisite dose of Progesterone but also ensure an adequate proliferative stimulus to the endometrium in those women who are not producing sufficient endogenous oestrogen.

Dosage consists of 4 tablets of Menstrogen daily on each of 5 successive days, dividing the dose over the 24 hours (one tablet every 4 hours).

The treatment may require to be repeated every 4 weeks for a few months. Each monthly course should be followed by a "period" in 3 to 5 days.

If treatment fails to induce a menstrual flow, the case should be investigated further.

Menstrogen Tablets are produced by Organon Laboratories Ltd. of London and are available in tubes of 20 and bottles of 60 from the South African distributors, Keating's Pharmacy Limited, P.O. Box 256, Johannesburg.

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VAN DIE REDAKSIE

HEMOFILIE: MODERNE BEGRIPPE

Hemofilie is lank reeds bekend as 'n oorgeërfde bloeisiekte van manlikes, met 'n verlengde bloedstollingstyd, as die mees merkwaardige laboratorium-abnormaliteit. Onlangse werk waartoe 'n Suid-Afrikaanse kollega belangrike bydraes [2, 4, 5] gelewer het, het dit vir ons nodig gemaak om ons idees omtrent hierdie siekte ietwat te heroriënteer.

Een van die misteries van die siekte was die afwesigheid van homosigote hemofiliese vroulikes. Volgens algemeen aanvaarde menings word die siekte as 'n resessiewe geslagsverbonde karakter oorgeërf, met die geen of geene verantwoordelik vir die oorerwing op die X-chromosoom aanwesig. Aangesien die man slegs een X-chromosoom het, sal hy simptome ontwikkel as hierdie chromosoom aangetas word. By die vrou, aangesien die aangetaste X-chromosoom die resessiewe geen dra, sal geen simptome ontwikkel nie, hoewel sy die siekte aan haar afstammelinge kon oordra. As sy 'n dubbele dosis van die gebrek sou ontvang dan behoort sy teoreties aan die simptome van die siekte te ly. Dit kan slegs gebeur as beide haar ouers die siekte kon oordra, d.w.s. as haar vader 'n bloeier en haar moeder 'n draer was.

Ondanks veelvuldige teenoorgestelde bewerings, was Wintrobe [1] in staat om te sê: ‚Geen egte geval van 'n vroulike bloeier is bekend nie.' Dit was beweer dat 'n dubbele dosis van die geen 'n dodelike uitwerking het deur die ontwikkeling van die embrio te verhinder; maar gedurende die laaste jaar het beide Merskey [2] en Israëls *et al.*[3] aanneembare voorbeelde van homosigote hemofiliese vrouens bekend gemaak. Dit skyn dat hulle skaarsheid toegeskrywe kan word aan die seldsaamheid van die paring om so 'n kroos voort te bring.

Die tweede toeverlaat van die diagnose, die verlengde tyd van stolling, is ook onlangs bestorm. Dit word dikwels gesê dat die wisseling in die tyd van stolling na normaliteit gedurende sluimerende fases van die siekte voorkom. Dit mag of mag nie waar wees nie.[4] Nogtans is gevalle van onbetwyfelbare hemofilie onlangs beskrywe waarby 'n normale tyd van stolling 'n permanente karaktertrek was; en soortgelyke laboratoriumbevindings was by ander aangetaste lede van dieselfde families, selfs in verskillende geslagte,[5] aanwesig. In die geheel was die siekte effens ligter as die gewone tipe van hemofilie, maar by

1. Wintrobe, M. M. (1951): *Clinical Hematology*, 3e druk. Londen: Henry Kimpton.
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3. Israëls, M. C. G., Lempert, H. en Gilbertson, E. (1951): Lancet, **1**, 1375.
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EDITORIAL

HAEMOPHILIA: MODERN CONCEPTS

Haemophilia has long been recognized as an inherited bleeding disease of males with a prolonged coagulation time of the blood as the most striking laboratory abnormality. Recent work (to which a South African colleague has made important contributions [2, 4, 5]) has made it necessary for us to re-orientate our ideas about this disease to some extent.

One of the mysteries of the disease has been the absence of homozygous haemophilic females. According to generally accepted genetic views, the disease is inherited as a sex-linked recessive character, the gene or genes responsible for its inheritance being contained on the X-chromosome. Since the male has only one X-chromosome, he will develop symptoms if this chromosome is affected. In the female, as the affected X-chromosome carries the recessive gene, no symptoms will develop although she will remain capable of transmitting the disease to her descendants. Should she receive a 'double dose' of the defect she should theoretically suffer from the symptoms of the disease. This could only occur if both her parents had been capable of transmitting the disease, i.e. if her father was a haemophilic and her mother a carrier.

Despite numerous claims to the contrary, Wintrobe [1] was able to say: 'No authentic case of a female bleeder is known'. It has been suggested that a 'double dose' of the gene had a lethal effect by inhibiting the development of the embryo; but in the last year both Merskey [2] and Israëls *et al.*[3] have published acceptable examples of homozygous haemophilic females. It seems that their scarcity can be attributed to the rarity of the mating required to produce such offspring.

The second sheet-anchor of the diagnosis, the prolonged coagulation time, has also recently been assailed. It is frequently stated that fluctuations in the coagulation time towards normal occur during quiescent phases of the disease. This may or may not be true.[4] But cases of undoubted haemophilia have recently been described in which a normal coagulation time was a permanent feature; and similar laboratory findings were found in other affected members of the same families even in different generations.[5] The disease, on the whole, was a little milder than the usual type of haemophilia, but in quite a

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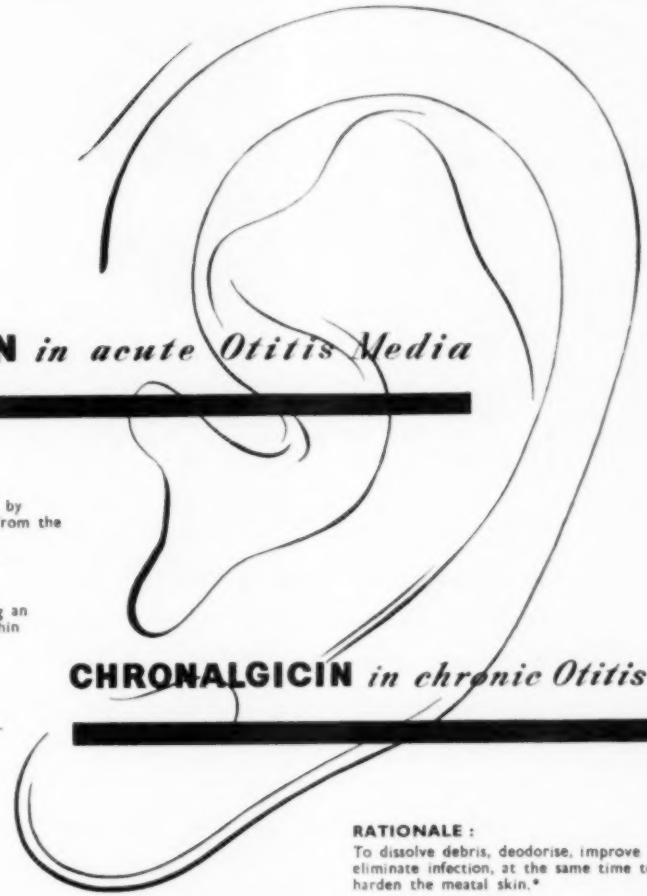

AURALGICIN *in acute Otitis Media*

RATIONALE :
To shrink inflamed mucosa and, by osmosis, establish full drainage from the middle ear.*
To eliminate pain and infection.

RESPONSE :
Auralgicin is capable of aborting an attack of acute otitis media within 24 to 36 hours.

REFERENCE :
*Reid, W. Ogilvy, Brit. Med. J. 1. (1946) 648.

CHRONALGICIN *in chronic Otitis Media*

RATIONALE :
To dissolve debris, deodorise, improve drainage and eliminate infection, at the same time to dry and harden the meatal skin.*

RESPONSE :
Improvement is noted early, but treatment may be necessary for some weeks before activity ceases or dry ear results.

REFERENCE :
*Reid, W. Ogilvy, Brit. Med. , 1. (1946) 648.

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heelparty van die pasiënte het 'n sindroom van aansienlike erns ontstaan en sterfgevalle aan die siekte het voorgekom.

Vir alle praktiese doeleindes bly die welgevestigde diagnostiese maatstawwe egter staan. Hemofilie is nog 'n siekte wat tot manlikes beperk is en in die oorgrootte meerderheid van gevalle is die tyd van stolling verleng. Die algemeenste oorsaak van 'n normale stollingstyd by hemofilie is verkeerde tegniek. Meeste van die metodes om die stollingstyd van kapillêre bloed te meet is onderhewig aan growwe foute, hoewel die Dale en Laidlaw-tegniek, indien sorgvuldig gebruik, waardevolle inligting mag inhou.[4] Voorkeur moet gegee word aan aarbloed, maar ook hier moet die tegniek foutloos wees as die resultate hoegenaamd iets moet beteken.

Gelukkig is hemofilie 'n siekte wat kliniès maklik erkenbaar is. As 'n pasiënt sê dat hy 'n bloeier is, is dit 'n goeie reël om hom so te beskou, wat die laboratoriumbevinding ookal mag wees. As sy bloeisiekte op die hemofiliese wyse oorgeërf is, en veral as dit verbind is met gewrigsbloeding, is dit feitlik seker dat hy hemofilie het. As die laboratoriumbevindings nog met die kliniese diagnose bots, behoort die pasiënt na 'n spesiale sentrum verwys te word waar sommige van die nuwer toetse vir hemofilie uitgevoer kan word.

number of the patients a syndrome of considerable severity resulted and deaths from the disease occurred.

For all practical purposes, however, the well-established diagnostic criteria remain. Haemophilia is still a disease confined to males and in the vast majority of cases the coagulation time is prolonged. The commonest cause of a normal coagulation time in haemophilia is faulty technique. Most of the methods of measuring the coagulation time of capillary blood are liable to gross errors, though the Dale and Laidlaw technique may give valuable information if done carefully.[4] The use of venous blood is to be preferred but here, too, the technique must be flawless if the results are to mean anything at all.

Fortunately haemophilia is a disease which it is easy to recognize clinically. If a patient states that he is a 'bleeder', it is a good rule to regard him as such whatever the laboratory findings may be. If his bleeding disease is inherited in the haemophilic way and especially if it is associated with haemarthroses, it is virtually certain that he has haemophilia. If the laboratory findings are still in conflict with the clinical diagnosis, the patient should be referred to a special centre where some of the newer tests for haemophilia can be carried out.

AN ANALYSIS OF 1617 CONSECUTIVE BIRTHS

AT ST. MONICA'S HOME, CAPE TOWN

ETHEL BARROW, M.B., CH.B. (LIV.), D.P.H. (CAPE TOWN)

Cape Town

The infants in this series were born during the period February 1947 to March 1950 in the old premises of St. Monica's Home, Cape Town. This covers approximately the last three years of work in the Home before the move to the new hospital in Lion Street. The old building at 182 Bree Street was originally a general store and was adapted to its use as a Maternity Home in April 1917 when it became the first training school for non-European midwives in the Union of South Africa. In 1919 the first antenatal clinic in Cape Town was opened there.

A brief description of the place is necessary in order to present the difficulties under which the staff had to work. The accommodation was inadequate and unsuitable. The roof leaked, the walls were damp and the place was rat-infested. The so-called nursery measured 12 × 13 ft., and was a glorified passage between a small room where patients' meals were served and waiting mothers were bathed, and a corridor leading to the ward. There were two windows measuring 3 × 5½ ft. and 3 × 4 ft. respectively. In winter the temperature was freezing and in summer it often rose to 90° F or more. Normally it contained about 14 cots placed touching each other in two rows. At times, however, as many as 26 infants were housed here and on these occasions baskets were placed on the floor under the cots. There were no facilities whatsoever for isolation.

The staff consisted of a European matron, 3 European sisters, 4 non-European staff nurses (trained midwives) and trainees. Most of the infants were delivered under the supervision of the staff nurses. No anaesthesia was used except in the forceps deliveries, and occasionally in other difficult deliveries.

During these three years 1,617 women were confined, but 21 were twin pregnancies, bringing the total number of infants born up to 1,638; 1,408 were of mixed origin, the large majority (1,317) Cape Coloured, 87 Malays, 4 St. Helenans. The Malays have been grouped with the Cape Coloured as a pure Malay is rare, the distinction being now chiefly one of religion, although Malay blood may predominate. Often a patient would call herself Malay when actually she was Cape Coloured married to a Malay. The remaining 209 belonged to various Native tribes. Including all races, 907 were unmarried, i.e. well over half.

The patients came from all parts of Cape Town and outlying suburbs with a few from country districts. Some belonged to the very poorest classes, while others came from the higher income groups. All types of cases were taken, including forceps deliveries and toxaemias of pregnancy, but excluding those requiring Caesarean section. The usual period of stay in the Home after the birth of the infant was 10 days. In many cases, however, the mother and child (sometimes the child only) were kept in longer. This was especially so in the case of premature birth. No infant was discharged unless its condition was satisfactory, except in cases where transfer to a general hospital was necessary.

The majority of cases attended the ante-natal clinics run by the Home and many of these were referred to St. Monica's from the ante-natal clinics of the Municipality of Cape Town. Therefore some benefited from the facilities which these clinics supply, such as free dinners, vitamin and iron therapy, and treatment for venereal disease and toxaemias of pregnancy. The general standard of nutrition, however, could be regarded as low, as most of the cases came from the lowest income groups; 89 were emergency cases, i.e. cases which had not attended the ante-natal clinic attached to the Home, but the greater portion of these had attended clinics elsewhere; 78 infants were born before arrival in the Home.

BREAST FEEDING

Satisfactory breast feeding was aimed at and of 1,565 infants discharged all except 54 were entirely on the breast (96.5%). A further 20 were partially breast fed and only 34 were completely off the breast. Of these, the condition of the mother was responsible for suppressing lactation in 17 cases; infection, 8; puerperal insanity, 2; suspected carcinoma of the breast, 1; hepatitis, 1; toxaemia of pregnancy, 1 (twins); severe eczema of the nipples, 1. Five infants were removed to hospital, excluding 3 who died there. Of the 5 infants it is possible that breast feeding was re-established on discharge in 4 of them. Six extremely premature infants, kept in the home after the discharge of the mothers, and two unrelated twins, whose mothers had insufficient milk for both, were bottle fed; so that only 4 mothers were unable to feed their infants because of complete failure of milk supply.

WEIGHT

In estimating the average birth weight only those of mixed origin were taken and the Bantu races were not included. Excluding twin pregnancies and stillbirths, 1,206 infants weighed over 5½ lb. each. A further 69 infants, however, weighing between 5-5½ lb. were considered full term as far as length and period of gestation were concerned. Taking the group of 1,206 infants weighing over 5½ lb., the average birth weight was 7 lb. 1 oz. Including the 69 infants considered otherwise full-term the average weight was 6 lb. 14 oz. These weights compare favourably with birth weights in London in 1938-1939, 1941 and 1942, in singletons, which were 7 lb. 4 oz., 7 lb. 2 oz. and 7 lb. 3½ oz. respectively. Recently, Woodrow and Robertson, in 1,000 cases taken from the records of Maternity Hospitals in Cape Town, found the average birth-weight in Coloured infants was 7 lb. 5 oz. and in European infants 7 lb. 11 oz. The largest infant in the St. Monica's series weighed 10 lb. 12 oz.

It is frequently stated that Negro infants have an average birth weight which is less than that of white infants. Brown *et al.* have suggested that the definitive weight for prematurity in the Negro race should be set at 5 lb. 3 oz. rather than 5 lb. 8 oz. because the full-term infants of white women are of relatively greater weight than those of Negroes. They found from their data that the lowering of the upper limit was justified. They mention that several investigators have found that the gestation period is shorter in the Negro than in the European. Taback also considers that 5 lb. 3 oz. would be a more logical upper limit of birth-weight for prematurity in the Negro. It is debatable whether this yardstick should be applied to our non-European population of mixed origin. Investigation on a much larger scale is necessary.

STILLBIRTHS AND MORTALITY

Of 1,638 births, 42 were stillborn and 31 were known to have died in the first 28 days of life, i.e. in the neonatal period. Because it was impossible to trace all the discharged infants, true neonatal figures could not be compiled, although no infant was discharged unless or until its condition was thought to be satisfactory. In view of this, statistics here cover the first 10 days of life only, and during this period 27 infants died—a rate of 16.9 per 1,000 live births; 15 (55%) of these infants died during the first 24 hours (9.4 per 1,000 live births). The Bureau of Medical Economic Research of the American Medical Association in a recent Bulletin (73A) expressed the opinion 'that it is possible to foresee the time when we would wish to discard the one-month concept in the neonatal rate and consider a time period of only one week'. In 1940 in the U.S.A. 83.3% of neonatal deaths occurred during the first week of life; 50% of all the deaths of the first month were in the first day, 13.9 per 1,000 live births. It seems desirable, therefore, that more statistics should be available for the few days following birth, as most of the hazards of labour and adjustments to extra-uterine life take place during this period.

CAUSES OF DEATH

Table I shows the weight distribution of 27 infants who died in the first 10 days. Nineteen in all fell into the premature group and 11 of these died in the first 24 hours.

TABLE I: WEIGHT DISTRIBUTION (TOTAL BIRTHS, STILLBIRTHS, DEATHS, ETC.)

Birth Weight	*Still-births*	*Deaths*		*No Autopsy*	*Total No. of Cases*
		First Day	*First 10 Days*		
Under 1,000 gm. (approx. 2 lb.)	6	2	2	2	8
1,000–1,500 ,, (,, 2–3½ lb.)	2	4	5	3	12
1,500–2,000 ,, (,, 3½–4½ ,,)	8	2	4	1	44
2,000–2,500 ,, (,, 4½–5½ ,,)	6	3	8	1	150
Over 2,500 gm. (,, 5½ ,,)	20	4	8		1,424
Total	42	15	27	7	1,638

TABLE 2: CAUSES OF DEATH IN 27 INFANTS

	Full time	*Premature*	*Total*
Anoxia	2	6	8
Intracranial haemorrhage	2	2	4
Congenital syphilis ..	1	1	2
Developmental defects ..	—	2	2
Infection	—	1	1
Haemorrhage (trauma) .	3	—	3
Prematurity only	—	6	6
No cause found	—	1	1
Total	8	19	27

Autopsies were performed on 20 of the 27 infants (74%). In Table 2 are listed what were thought to be the principal causes of death. The 6 infants under the heading 'Prematurity Only' were not examined *post mortem*. All weighed less than 3½ lb. at birth (Table 1), 3 were born before arrival in hospital, 2 were twins (unrelated) and 5 died within the first 24 hours. Autopsy may have revealed a more definite cause of death. Clinically, all showed signs of anoxia. There was no serological evidence of syphilis in the mothers, nor was there in the seventh case, in which no autopsy was performed. This infant was a twin weighing 4 lb. 12 oz. who died in a general hospital on the eighth day, following the removal of blood-stained fluid per vaginam, a tentative diagnosis of haematometra having been made.

In 9 of the mothers of the remaining 20 infants, the Wassermann reaction was positive ante-natally and 7 were still positive post-natally. One mother had clinical signs of syphilis at the time of her confinement. Her infant weighed 4 lb. 8 oz. at birth and died on the seventh day in spite of concentrated Penicillin treatment. At autopsy no spirochaetes were found but changes in the liver and other organs were similar to those found in congenital syphilis. One other case showed syphilitic lesions at autopsy. Where prematurity, anoxia and evidence of syphilis in the mother were all present in the same case, classification was based entirely on the autopsy reports. In a Ministry of Health Report (No. 94) it is stated that 'the spirochaete cannot always be demonstrated in fresh syphilitic infants, and one of the infants in which spirochaetes were not found was a twin of a macerated foetus in whose organs they were specially numerous'.

In 4 cases there was no history of any ante-natal treatment; one case had received 4 injections of arsenic and another 3 injections. Three cases had been treated for a few weeks ante-natally. In no case could the treatment be regarded as adequate. Seven of these infants were premature and it is possible that syphilis may have been the cause of the prematurity.

The 3 cases of haemorrhage were as follows:

1. Massive bleeding from the base of the umbilical cord on the third day. A patent vessel was found which corresponded to the umbilical vein.

2. Extravasation of blood over the entire scalp under the sub-aponeurotic layer and coming from a ruptured cephalhaematoma over the right parietal bone.

3. Massive haemorrhage into the peritoneal cavity from a ruptured haematoma on the under surface of the liver.

Only one infant died from demonstrable infection and this was a case of bronchopneumonia. In addition, in this case the Wassermann reaction was positive ante-natally and post-natally.

Of the 4 other cases known to have died in the first month and not included in the 27 deaths above, 3 weighed 2 lb. 9 oz., 2 lb. 15 oz., 4 lb. 2 oz., respectively, at birth. Autopsy was performed on the 2 infants weighing 2 lb. 15 oz., and 4 lb. 2 oz., but no cause of death was found. The fourth case was a twin who died from a tracheo-oesophageal fistula.

The importance of prematurity as a factor in neonatal death is again demonstrated in this study. Including twin pregnancies just over 12% of the infants born alive were premature and 70.4% of the deaths in the first 10 days of life were in premature infants. Macgregor in her series of 618 neonatal deaths, found that 70.5% were in cases of premature birth and slightly over 10% of all live births in the hospital were premature.

Potter and Adair give mortality figures of the Chicago Lying-in Hospital in 1947 for the first 10 days of life in infants weighing over 1,000 gm. The rate was 11.8 per 1,000 live births, as compared with 15.5 per 1,000 in this series. It is significant, however, that only 5.9% of their cases fell into the premature group and 65% of the deaths were in this group.

STILLBIRTHS

There were 42 stillbirths, a rate of 26.2 per 1,000 total births. In England and Wales in 1945 the stillbirth rate was 28 per 1,000 total births, but different areas show varying rates ranging (1939-1941) from 30 in Greater London to 43 in Manchester and Salford and 46 in Wales. In the United States of America in 1945 the still-birth rate for white children was 21.4 per 1,000 live births; for non-white, 42.0.

Analysis of the causes was unsatisfactory as autopsy was obtained in only 6 cases. In Table 3 are listed what were thought to be the main causes. Nineteen of the infants were macerated. Prematurity was present in 22 cases, but prematurity in itself cannot be regarded as a cause of still birth. The autopsy findings were of very little assistance, as 4 cases were macerated. Signs of anoxia were present in the 2 remaining cases. Serological evidence of syphilis was present in 12 of the mothers. Seven of these infants were macerated and 6 were premature. In at least 9 cases there appared to be no reason for foetal death apart from syphilis.

TABLE 3: CAUSES OF STILLBIRTH IN 42 CASES

Cause	*Term*	*Premature*	*Total*
Anoxia			
Complications of labour or delivery	8	6	14
Syphilis	3	6	9
Toxaemia of pregnancy ..	3	1	4
Abnormal placenta ..	—	1	1
Developmental defect ..	1	—	1
Undetermined	5	8	13
Total	20	22	42

Macgregor (1946) studied 618 neonatal deaths and 453 stillbirths in Edinburgh (all submitted to autopsy), and concluded that the chief causes of death, in order of importance, were birth injury, anoxia, infection and malformations. Potter and Adair (1943) reported on 559 neonatal deaths and 614 stillbirths at the Chicago Lying-in Hospital over a period of 10 years (autopsy in 81%), and found that anoxia, birth injury and malformations in that order were the chief causes. They found infection responsible in 4.7% only, as compared with 19% in Macgregor's series. Syphilis was found in 2 cases (0.2%). Macgregor states that syphilis caused a small number of still births and very few neonatal deaths, but she does not quote figures. She believes that, if a syphilitic infant is born alive, it usually survives the neonatal period. Potter and Adair consider that infection plays a minor role as a cause of death in the first few weeks of life, as compared with its importance in later life. Macgregor found that infection was more likely to occur after the first few days.

Table 4 shows mortality and stillbirth rates from different sources.

Twenty-two of the premature infants were stillborn and 19 died in the first 10 days. Prematurity was thus present in 52.4% of the total stillbirths and 70.3% of the deaths. Table 5 shows the percentage of survivals and in these figures are included the 4 infants mentioned above, who were known to have died in the first month. Although these figures are not entirely comparable with others because of the time factor, a large number of the premature infants, especially those in the lower weight groups, were kept in hospital much longer than 10 days, many reported back or were followed up, and no infant was discharged unless its condition was considered satisfactory. In the lowest weight groups (1,500 gm. and under) more than half were born before arrival in hospital, which reduces their chances of survival enormously.

Crosse's report based upon the figures for the City of Birmingham shows that the neonatal mortality rate for immature infants for 1943 was 278 per 1,000. The rate in this series was 120 per 1,000 and although not strictly comparable, as shown above, the difference is so great that it suggests that the lower weight groups in this series

TABLE 4: MORTALITY AND STILLBIRTH RATES FROM DIFFERENT SOURCES

	Stillbirths	*Mortality Rates per 1,000 Live Births*			
	Per 1,000 Total Births	*First Day*	*First 10 Days*	*First Month*	*First Year*
St. Monica's Home (1947–50)	25·6	9·4	16·9* 15·7† 11·8†		
Chicago Lying-in Hospital (1947)					
England and Wales (1942)	33			27	51
Scotland (1942)	38			34	69
United States of America (1945)	23·9‡	11·2		24·3	38·3
Whites	21·4‡				
Non-Whites	42·0‡				
City of Cape Town (1949–50)					83
Europeans					29·5
Non-Europeans					101·5

*Total mortality. †Infants weighing 1,000 gm. or more. ‡Per 1,000 live births.

PREMATURITY

In this group are included all infants weighing 5½ lb. (2,500 gm.) or less, and of 28 weeks' or more maturity since conception in accordance with the standard in international use.

Including multiple births, 214 infants (13.06% of the total births) belonged to this group, 188 being of mixed origin and 26 Bantus. Crosse (1945) who analysed the figures for the City of Birmingham in 1943 found that 6.3% of total births (living and still) were immature. Analysis of cases admitted to the Aberdeen Maternity Hospital (Baird, 1945) showed that labour terminated prematurely in 8.3%. In Baltimore City in 1949, 7.4% of white infants were premature as compared with 12% Negroes.

It would appear that the incidence of prematurity in this series is high and the question of a racial element should perhaps be considered. Taback found that by taking 5 lb. 3 oz. as the limit, the differential in percentage prematurity between races is eliminated. Using this limit the incidence here falls to 9.5%.

TABLE 5: SURVIVAL RATE OF PREMATURE INFANTS ACCORDING TO BIRTH WEIGHT

Birth Weight	*Holt and McIntosh* (1940)	*St. Monica's Home*
	%	%
1,000 gm. (approx. 2 lb.)	5	0
1,000–1,500 gm. approx. 2 –3½ lb.)	33	30
1,500–2,000 „ („ 3½–4½ „)	75	86
2,000–2,500 „ („ 4½–5½ „)	90	94

have a greater degree of maturity than the European infant in the same group.

Analysis of groups of premature births shows that a cause of prematurity can be demonstrated in only about 50% of cases (Ministry of Health No. 94), and in this series in only 40% was there a demonstrable cause. There were 45 cases of syphilis, 24 multiple births, 11 maternal toxaemia, one foetal malformation, 2 cases of accidental haemorrhage, one abnormal placenta and one placenta

praevia. For the rest no definite cause could be discovered, but poor nutrition may play a part and again there may be a racial difference in the degree of maturity.

SYPHILIS

Excluding twin pregnancies syphilis was present in the mother in 23.7% of premature births, whereas only 8.2% of the mothers of full-term infants were syphilitic. The incidence of prematurity in syphilitic mothers was 27.8% and in non-syphilitic mothers it was 10%. The total incidence of syphilis including all births was 10%; 34.6% of the infants who died and 29.2% of the stillbirths were born to syphilitic mothers.

TOXAEMIA OF PREGNANCY

Statistics in this group vary considerably because of different classifications of toxaemias of pregnancy.

In this series all patients with a blood pressure of over 120/90 mm. Hg were included according to Baird (1950). Of a total of 1,529 cases with available ante-natal records, there were 86 cases of pre-eclampsia and 6 cases of eclampsia, a total incidence of 6%. One eclamptic patient died an hour after admission and before delivery. This was the only maternal death in the Home during the period under review. One other patient died after removal to another hospital from arsenical encephalopathy.

The incidence of toxaemia in hospital practice in England varies from 3.5%-10% of pregnancies and is rarely below 5%. It is frequently stated that toxaemias of late pregnancy are the most important of the maternal diseases which affect ante-natal, natal and neonatal mortality, and many statistics are available to support this view. In America Wellen (1940) reported a foetal mortality rate of 30.8% and Scott (1940) a mortality rate of 27.2%. In England the figures quoted for Manchester are 29.2%, Newcastle 46.9%, London 25.6%, and Liverpool 21.5%. In England the total loss of child life in eclampsia is between 40%-60%.

In this study 4 infants were stillborn and the infant of one toxaemic mother died on the third day, but the mother was also syphilitic, and signs of congenital syphilis were found at autopsy. Even including this case, the total foetal mortality rate was only 5.5% (during the first 10 days). Of the 5 cases of eclampsia who were delivered, 2 infants were stillborn and 4 (one twin pregnancy) survived. On discharge 56 infants had either regained their birth weight or weighed more than at birth. Potter and Adair believe that there is no evidence that toxaemia affects an infant after delivery except as it may have contributed to a premature or complicated birth. The incidence of prematurity here was 12.1%, i.e. not higher than in the group of infants as a whole, and 5.4% of the premature infants were born to toxaemic mothers. Table 6 shows the frequency of prematurity amongst toxaemic mothers, from various centres. They differ widely because the severity, methods of treatment and the classification of toxaemia vary in different centres. The low incidence recorded in this series may be explained partly by the high rate of prematurity from other causes and because about a quarter of the cases included could be considered mild in character.

RH INVESTIGATIONS

Blood examinations for Rh grouping were carried out on a great many of the mothers but exact figures are not available because, owing to a faulty filing system, it was impossible (without a great deal of labour) to separate the in-patients from the out-patients. However, 15 women were found to be Rh-negative, 8 were primipara and the rest gave birth to normal infants. Only one case of haemolytic disease was encountered and that proved to be a Fisher's E/e incompatibility.

ILLEGITIMACY

The mortality rate in the married group was 0.1% higher than in the unmarried mothers and the still-birth rate 0.6% higher in the unmarried mothers. The difference is so slight that it appears that in this country illegitimacy is not a significant factor in foetal and neonatal mortality as it is in European countries.

INFECTIONS

Working under such unfavourable conditions one would have expected a high rate of infection, but this was not so. One infant died from bronchopneumonia. There were 2 cases of salivary gland infection, one involving the left submaxillary gland and the other the lingual gland. Both cases recovered completely. Eye infections occurred in 24 cases but were usually of a mild nature. *N. gonorrhoeae* was found in 2 cases and for the rest diphtheroids and staphylococci were the most common organisms involved. Rarely was there more than one case at a time in the nursery, and usually only one eye was involved. Skin lesions included erythemas, pustules, buttock rashes and pressure sores, but they were infrequent and not severe. There were a few cases of thrush. Cross-infection rarely occurred. There were 2 cases of syphilitic pemphigus and these cleared up with concentrated Penicillin treatment. Except in the case of bronchopneumonia, no evidence of infection apart from syphilis was found at autopsy.

COMMENT

It would appear from the figures obtained in this analysis that the non-European infants (including those of mixed origin and Bantus) have as good a chance of being born alive or surviving the first few days of life as infants born

TABLE 6: FIGURES FROM VARIOUS CENTRES SHOWING THE INFLUENCE OF TOXAEMIA ON THE INCIDENCE OF PREMATURITY

Hess *et al.* (1934)	U.S.A.)	16%	premature infants born to toxaemic mothers.
Clifford (1934)	,,	30%	premature infants born to toxaemic mothers.
Massey (1940)	,,	25%	premature infants born to toxaemic mothers.
Belfast		49%	premature infants born to toxaemic mothers.
London		30%	premature infants born to toxaemic mothers.
Liverpool		21%	premature infants born to toxaemic mothers.
Dublin		16·6%	premature infants born to toxaemic mothers.

elsewhere. The stillbirth rate, although high, compares very favourably with other figures quoted. The high rate of prematurity is partly due to syphilis. Poor nutrition in the mother may also be a factor. It is debatable whether the upper limit of weight in premature infants should be reduced in the case of the non-European races. It is interesting that, in spite of poor nutrition, successful breast feeding was established in 96.6% of cases. Whereas congenital syphilis in America and Great Britain has been almost eliminated, it still plays an important role here as a cause of stillbirth and death. Too often the mother attends the ante-natal clinic for the first time too late in her pregnancy.

The infant mortality rate in Cape Town amongst non-Europeans for the period 1948-49 was 110.8 per 1,000 live births; in 1949-50, 101.4. The European figures for these periods were 29.2 and 29.56. The chief wastage in infant life in the non-European therefore occurs after the first few days of life, whereas in other groups more than half the infants dying in the first year are lost in the first month.

More figures for comparison are desirable, and larger series should be dealt with for more accurate results. Better results are probably obtained in hospital than in domiciliary practice, although these facilities (which were at a minimum in the cases under review) are offset by a higher incidence of difficult labours and toxaemic cases.

Local authorities in this country would do well to include either neonatal rates or rates for the first few days of life in their statistics in future.

SUMMARY

An analysis has been made of 1,617 consecutive births at St. Monica's Home, Bree Street, Cape Town, from February 1947 to March 1950.

Figures relating to birth weight, prematurity, still births, deaths, etc., are presented.

The chief causes of still births and deaths are discussed.

The incidence of prematurity and of syphilis was found to be high.

I would like to thank Dr. A. Simpson Wells for his permission to publish this article and for his helpful advice; also the Matron and staff of St. Monica's Home, who so readily co-operated at all times and, in particular, Sister Bright (who was in charge of the infants for most of the time). My thanks are also due to the Pathology Department of the University of Cape Town who performed the autopsies, and to Drs. Clegg and Finlayson for Rh investigations.

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BACTERIAL PNEUMONIA*

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The magnitude and complexity of the problem of pneumonia has remained a challenge since the earliest days of the Witwatersrand gold mines. The toll in human suffering and the cost to the mining industry can be appreciated from the fact that during 1948 on the City Deep Limited, out of a total of 63,141 working shifts lost, 25,882 were due to accident, and 37,259 due to disease; of the latter, 11,680 were due to pneumonia.

Maynard [1] stated that the clinical diagnosis of pneumonia among Native mine labourers presented grave, if not insurmountable, difficulties. In 1913, Sir A. E. Wright [2] issued his report on pneumonia. Lister made an extensive study of the pneumococcal serological reactions [3] and prophylactic inoculation of man against pneumococcal respiratory infections [4]; and with Ordman,[5] investigated the epidemiology and prevention of pneumonia.

Watkins-Pitchford and Allan [6] were puzzled by the significance of persistent rales in the lungs of apparently healthy Natives. Emanating from the desire to establish universal uniformity in diagnosis, it was agreed by Orenstein and Daubenton [7] that a case should not be classified as lobar pneumonia except in the presence of the following:

(a) Definite signs of consolidation of a lobe or portion of a lobe;
(b) Crepitant rales in some stage of the disease;
(c) Crisis, pseudo-crisis or rapid lysis, except when complications are present.

No one would doubt that an acute respiratory infection fulfilling all the above criteria would be a case of lobar or partial lobar pneumonia. However, since the use in 1938 [8] of chemotherapy in the treatment of pneumonia on the mines, and its subsequent widespread use in the treatment of acute febrile respiratory infections, the difficulty in clinical diagnosis of pneumonia has been much increased and, in the adoption of the above or any similar standardization, it has become apparent that cases are

* The References will be published at the end of the concluding part of this article.

being missed completely. This is either because the disease, during its course, may exhibit indefinite or no physical signs of consolidation and no crepitations or, more frequently, that the disease process is arrested by chemotherapy before any definite clinical signs have become apparent.

It is a matter of individual opinion whether every case of suspected acute respiratory infection should be treated immediately with chemotherapy or not. By expeditious therapy in an acute respiratory disease the patient is spared the discomfort of delay in treatment and in the majority of cases is assured a speedy return to health, without a precise diagnosis being made. It then becomes necessary to conduct a more diligent examination in quest of a precise diagnosis in only those cases in which the hoped for therapeutic result is not forthcoming.

Delay in treatment while in quest of a diagnosis introduces its difficulties. Firstly, interpretation of the frequently insignificant signs suggesting the diagnosis may be confusing; secondly, admirable restraint may have to be shown in withholding chemotherapy pending diagnosis, although the patient is febrile; and thirdly, on humanitarian grounds, strong disagreement may be expressed with those who delay chemotherapy as a matter of routine until a definite diagnosis is made. Certainly for statistical purposes a precise diagnosis is essential.

The present study was prompted by the realization that the altered clinical course of bacterial pneumonia, since the advent of the widespread use of chemotherapy, remained ill-defined, and is an attempt to define the nature of the clinical course of the disease as now encountered.

The material used consists of cases of bacterial pneumonia admitted to one medical ward of the City Deep, Central Mine Native Hospital, during the months of November and December 1948, and January and February 1949. Fifty-two cases were analysed and are here presented. A larger series of 730, the total number of cases of pneumonia diagnosed during 1948, is also briefly discussed.

CLASSIFICATION

A modification of Finland's classification[9], although somewhat unwieldy, helps to bring bacterial pneumonia into perspective in the whole field of pneumonia.

1. *Bacterial Pneumonias.* Pneumococcal, streptococcal, staphylococcal, Friedlander's, *H. influenzae* and other bacterial pneumonias produced by meningococcus, *Micrococcus tetragenus, Neisseria catarrhalis, B. anthracis, P. pestis, P. tularensis.*
2. *Mycotic Pneumonias.* Moniliasis, coccidiomycosis, aspergillosis, etc.
3. *Rickettsial Pneumonias.* Typhus, Rocky Mountain spotted fever, South African tick fever (tickbite fever).
4. *Known Virus Pneumonias.* Measles, smallpox, lymphogranuloma, lymphocytic choriomeningitis.
5. *Influenzal Virus Pneumonias.*
6. *Psittacosis, Ornithosis.*
7. *Pneumonias, presumably due to an unidentified virus.*

Firstly, it has been noticed that the bacterial pneumonias fall into 2 clinical types:

1. Pneumonia following directly on an upper respiratory infection: bronchitis, catarrhal cold, sinusitis, etc.
2. Pneumonia of sudden onset without any history or evidence of an upper respiratory infection leading up to the attack.[10]

Secondly, to demonstrate the variety of clinical pictures which this disease presents, further subdivision based on a combination of correlated clinical and radiological observations is presented:

1. Classical lobar pneumonia.
2. Partial lobar pneumonia.
3. Segmental pneumonia.
4. Central pneumonia.
5. X-ray negative pneumonia.
6. Progressing pneumonia.
7. Diffuse pneumonia.
8. Bronchopneumonia.

PATHOGENESIS

It is increasingly apparent that an upper respiratory infection, upper respiratory catarrah, bronchitis and actual pneumonia are all different stages in the natural course of a disease process. A case which to-day is a mild upper respiratory catarrah or a bronchitis, may to-morrow be a pneumonia; hence the diagnosis of an individual case may require to be revised from day to day.

It has been observed that in man iodized oil introduced into the nose during sleep can be found radiologically in the lungs next morning. It seems possible that infected mucus of nasal or bronchial origin, on trickling down into the lungs, may block a bronchus or bronchiole, cause an infected atelectasis with resultant pneumonic consolidation[10] (Fig. 1A). The organisms involved are usually mixed and vary in virulence, the degrees of upper respiratory catarrah or bronchitis and the resistance of the patient vary; hence it is not surprising that the lung changes and clinical pictures associated with them are diverse.

In pneumonia of sudden onset without an upper respiratory infection leading up to the attack, the clinical pictures and particularly the radiological appearances, can be correlated strikingly with the present trend of knowledge in the pathogenesis and pathology of pneumonia. Frequently, in cases with symptoms of about 12-24 hours' duration, the original focus of infection in the lungs can be identified radiologically as a shadow of about 1-2 inches in diameter, seen usually in a segment situated in the dorsolateral aspect of the lung substance, and those lesions which spread, do so rapidly until the process is arrested at the inter-lobar septum, part of, or a whole lobe, thus becoming involved (Fig. 1B).

This conforms with the concept that the lesion is an allergic reaction in an individual who has previously been sensitized by an organism. The patient contracts a mild respiratory infection, sinusitis, pharyngitis or bronchitis, which settles. With a second infection, organisms of adequate virulence gain access to the lung substance and, establishing themselves, elicit a focal allergic oedema.[11] This oedema fluid teeming with organisms, passes from one alveolus to the next through Kohn's pores and, by means of the movements of respiration, coughing and changes in posture, are driven along the bronchioles and smaller bronchi, effecting spread of the disease.

CLINICAL PICTURES

Age. The average age of the patients under consideration in this series was about 26 years.

1. *Pneumonia Following Directly on an Upper Respiratory Infection, Bronchitis, Catarrhal Cold, Sinusitis, Etc.* During routine radiological examinations, transient

shadows are occasionally detected in the lungs of Native labourers who either admit some recent slight cold or cough, or deny any such episode and in whom, on clinical examination, a few crepitations, rhonchi or nothing at all, may be detected. It seems that the explanation of these transient shadows lies in an infected atelectasis (Fig. 1A).

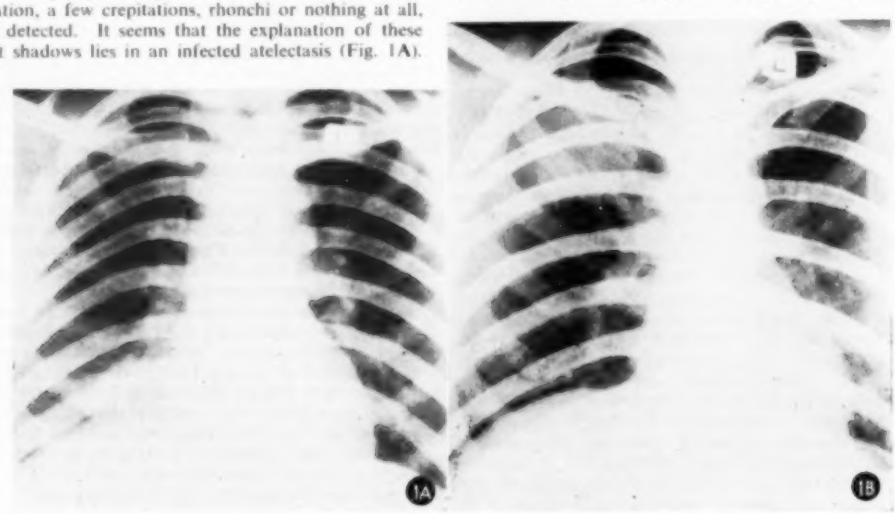


Fig. 1A. Segmental pneumonia at the right base, showing the fan-shaped shadow of infected atelectasis.
Fig. 1B. Segmental pneumonia, at the right apex, showing the circular shadow of an acute allergic response.

Also, in cases admitted to hospital with little more than a febrile common cold and related trivial catarrh, a chest radiograph may show shadows which can best be interpreted as localized areas of consolidation in the lungs, although the patients have none of the symptoms or signs customarily associated with acute pneumonia.

Further, cases may present with generalized body pains, headache, and perhaps an indication in the form of a gesture of some generalized discomfort or more localized pain in the chest. Examination reveals a mildly ill patient, with pyrexia, slightly increased pulse and respiratory rates, sputum in the form of a yellow mucus and rhonchi as the only adventitious sounds audible with a stethoscope. The signs elicited will substantiate only a diagnosis of bronchitis. Treatment with chemotherapy usually cures the attack rapidly. There are 3 points about this type of case:

1. Not infrequently a case which is only a bronchitis will be found, on radiography, to show a shadow of consolidation in the lung.
2. Rhonchi localized to one lobe only suggest the presence of a patch of consolidation in that lobe, even in the absence of more characteristic signs.
3. If resolution does not occur, or is delayed, a patch of dullness, bronchial breathing, crepitations or bloody sputum will indicate the sudden appearance of an area of consolidation.

2. *Pneumonia of Sudden Onset Without any History or Evidence of an Upper Respiratory Infection Leading up to the Attack.* This group of cases of pneumonia lacks the direct sequence of following straight on from a mild upper respiratory infection and would appear to arise from the action of bacteria with a more definite pneumotrophic action. There is usually a sudden onset of acute febrile illness, with symptoms of, perhaps, generalized pains, headache, with or without pains in the chest, increased pulse and respiratory rates, other than which initially there may be no signs in the chest or, if seen later, there may be early signs of consolidation.

CLINICAL SYMPTOMS

The onset is sudden, the patients being able to tell within a few hours the time of onset.

The mode of onset in 52 cases was as follows:

Localized pain in the chest: 30 cases.
Pain across the front of the chest: 9 cases.
Headache and pain in the chest: 8 cases.
Headache: 2 cases.
Pains all over the body: 1 case.
Headache, pain in the chest and pains all over the body: 1 case.
Abdominal pain: 1 case.

Thus, in 90% of cases, initial symptoms directly referable to the chest were present, leaving only 10% in which the initial symptoms were not in the chest. Although the majority of cases of bacterial pneumonia present with symptoms of pain in the chest, it is by no means diagnostic of pneumonia, for frequently cases present with pain in the chest and pyrexia and show no other symptoms, no signs, no radiological evidence of pneumonia and no leucocytosis at any stage of the disease.

CLINICAL SIGNS

It is realized that physical signs in themselves, even when taken in groups, are frequently not conclusive proof of any particular disease, and that to choose any one sign

as diagnostic of a disease is even less reliable, but a combination of any 2 of the physical signs below, if they can be elicited, are usually diagnostic:

i. *Blood in the sputum* occurs mixed in a froth, indicating the pulmonary origin. Streaking with blood is not nearly such a dependable sign.

ii. An area of *dullness* which develops with the disease and disappears with cure.

iii. A patch of *bronchial breathing* coming and going as the disease progresses to its height and subsequent cure.

iv. The diagnostic value of *crepitations* as a sign in a case is often difficult to assess, for crepitations are often heard for varying lengths of time in people who are fit and in chests which in every other respect are normal.[6]

v. *Vocal resonance and fremitus* are particularly valuable signs. Vocal resonance is a very sensitive, if not the most sensitive sign for detecting and localizing a patch of consolidation and is the last sign to disappear, remaining on in diminishing intensity until the lung is radiologically clear.

vi. *Diminished air entry* is the earliest audible physical sign detectable in acute pneumonia. It is often difficult to be sure of it as a sign, for the air entry varies normally in different regions of the chest; those regions which most consistently show the greatest air entry are the right apex, and left base posteriorly and laterally.

How much are we to rely on the readings of the temperature, pulse and respiration so mechanically charted by nurses and so carefully scrutinized by doctors? The records here discussed were all taken and recorded by a reliable member of the staff.

Temperature Records. Accurate readings can only be assessed when the thermometer has been left in the mouth for 10 to 15 minutes, the mere half-minute affair advocated by the manufacturers being quite inadequate.[12]

In this series the thermometer was left in the mouth for one minute. This means that the readings were probably low, but could be compared with each other in the same case and served as a comparison between cases.

Other sources of error are:

(a) Exposure of the patient to cold prior to admission;

(b) Inaccuracies in the thermometers in use;

(c) Inaccuracies in the recordings made by the observer.

Taking all these possible sources of error into consideration, interpretation of the significance of temperature readings must be made with an understanding of the circumstances in which the readings were taken and recorded.

Pyrexia. While realizing that there is an individual variation in normal temperature, pyrexia is here considered to be any reading above 98.4° F.

Temperatures were recorded twice daily and pyrexia was considered to have terminated at the first reading at which the temperature reached and remained permanently below 98.4° F. The duration of pyrexia ranged from no pyrexia at all to a period of 18 days. The average duration of pyrexia in this series of 52 cases was 3.04 ± 0.205 days (Fig. 2).

Maynard[13] in 1913 found, among 'Tropical' Natives, under similar living conditions at the Witwatersrand Native Labour Association's compound, the mean day of crisis to be 5.35 days and in those cases in which the temperature fell by lysis, the mean to be 7.46 days. Thus there has been a considerable reduction in the duration of pyrexia in the disease as encountered since the advent of chemotherapy.

Termination of pyrexia appears to coincide with the cessation of activity of the infection, for the patient suddenly feels and looks better and regains health from this point onwards.

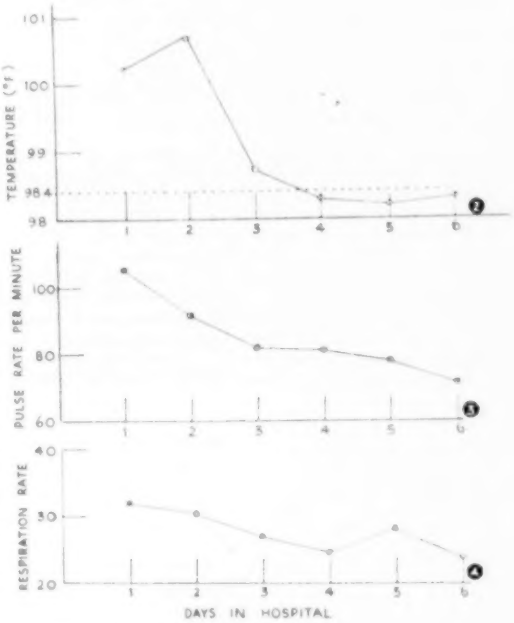


Fig. 2. Average temperature.
Fig. 3. Average pulse rate per minute.
Fig. 4. Average respiration rate per minute.

The Pulse. In contrast to temperature taking, the readings in pulse taking are more likely to be too high than too low and initial excitement may render the result misleading.[14] Real counting should not commence until the pulse has settled down to a steady performance, which may be as late as the third minute. The mean daily pulse rate on the first hospital day was 105 per minute, falling sharply to 82 per minute on the second day, with a more gradual fall to 71 per minute on the sixth day (Fig. 3).

Respiration. As in pulse taking, the errors in respiration records are predominantly emotional in origin. The mean daily records of the respiratory rates while in hospital showed the mean rate to be 32 per minute on the day of admission, and 23 per minute on the sixth day (Fig. 4). Thus, although the patients felt and looked well and wanted to be discharged from hospital, the majority of cases were discharged to convalescent gangs with respiration rates above the conventional normal of 14-18 per minute. This may be associated with the fact that most of the cases were discharged from hospital with radiological evidence of a lung lesion still present. The respiration rate settles to normal after the temperature and pulse, but it was surprising that the rates were still so high when the patients were discharged from hospital.

Pulse: Respiration Ratio. This ratio is normally 4 : 1. A comparison of Figs. 3 and 4 shows that the ratio for the first 6 days is in the neighbourhood of 3 : 1 (Table I):

TABLE I—PULSE: RESPIRATION RATIO

Day	1st	2nd	3rd	4th	5th	6th
Ratio	3·3:1	3·0:1	3·1:1	3·35:1	2·74:1	3:1

Sputum. The appearance of a bloody or rusty sputum is very strong evidence in favour of bacterial pneumonia. When other signs are suggestive it may be the sign which clinches the diagnosis. There are, however, many cases with bacterial pneumonia which do not show a bloody sputum. In this series of 52, bloody or rusty sputum was seen in 35 cases, yellow mucus in 13 and just froth in 4.

Total White Cell Count. The daily average white cell count varied directly with the temperature, and during its fall reached the 10,000 figure or lower, shortly after the temperature had reached 98.4° F. Counts were not done sufficiently frequently to assess the exact intervals (Fig. 5).

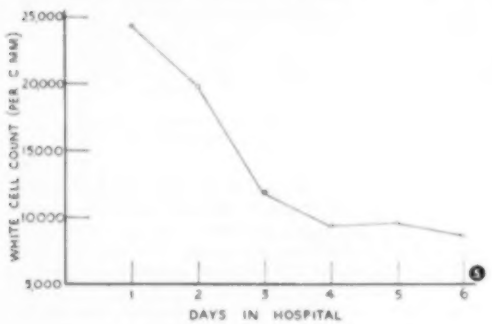


Fig. 5. Average white cell count.

Neither the total white cell count nor the temperature was a direct indication of the size of the lesion, for some of the smallest lesions radiologically had the highest white cell counts and temperatures.

The total white cell count was taken at the first opportunity, all on the first hospital day, with the exception of 4 taken on the second day.

White Cell Count (per c.mm.):	*No. of Cases.*
Under 5,000	Nil
6,000-10,000	8
11,000-15,000	9
16,000-20,000	10
21,000-25,000	12
26,000-30,000	4
31,000-35,000	5
36,000-40,000	3
41,000-45,000	Nil
46,000-50,000	Nil
51,000-55,000	1

Relationship Between Length of History, Length of Time in Hospital Before Sulphapyridine was Administered, and the Duration of Pyrexia in 52 Cases of Pneumonia.

In order to establish this relationship, 3 sets of information were considered:

1. Length of history of cases with pneumonia. This was the number of hours, from the onset of symptoms to the commencement of treatment with Sulphapyridine.
2. Length of stay in hospital in hours, before the administration of Sulphapyridine.
3. Duration of pyrexia, in hours, after admission to hospital.

In Fig. 6, the length of the case history is plotted below the line A—A, and the duration of pyrexia after admission and the delay after admission, before Sulphapyridine was administered, are plotted above the line A—A. This shows that there is absolutely no relationship between the

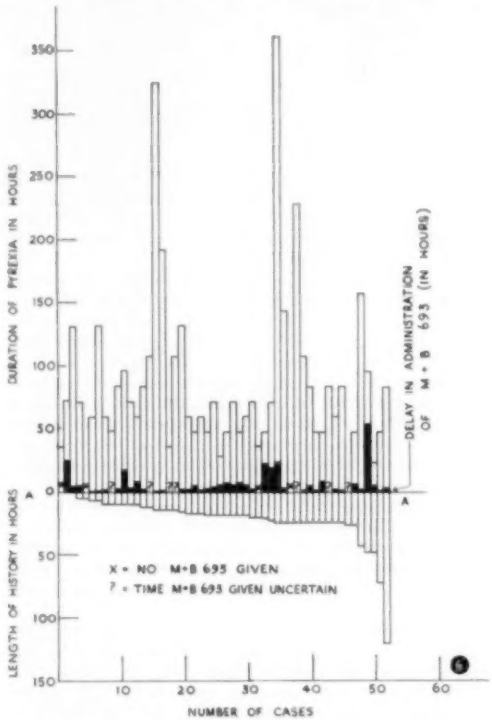
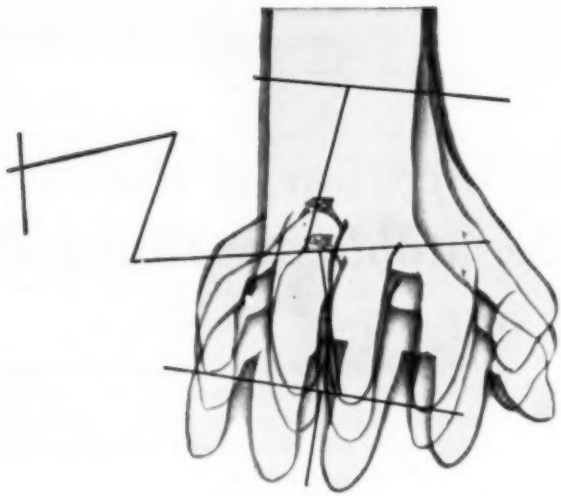


Fig. 6. Cases arranged in ascending order of length of history.

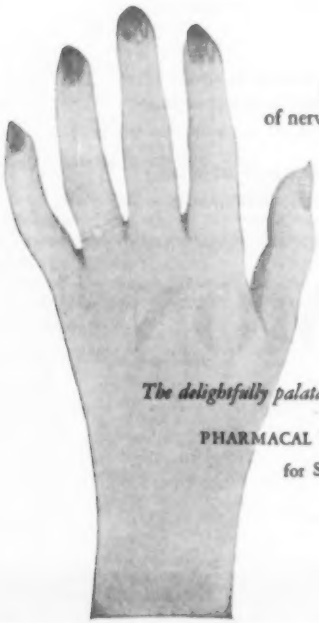
length of case history and the duration of pyrexia. In addition, it appears that the time before Sulphapyridine was given does not have any bearing on the duration of pyrexia, as some cases in which Sulphapyridine was delayed recovered quickly, while others in which it was administered immediately had a long pyrexial period.

Having decided that the length of case history has no bearing on the other results, the results in Fig. 7 were arranged in ascending order of periods, before Sulphapyridine was administered below the line A—A and the duration of pyrexia, in hours, in each corresponding case



"one nervous woman can give rise to more diverse, undiagnosed and undiagnosable complaints than a whole pathological ward."

Harding, T. S.: M. Rec. 160:198, 1947



For the many patients, especially women, who complain of nervous tension throughout the day and wakefulness during the night, ESKAPHEN B ELIXIR is an ideal preparation.

ESKAPHEN B ELIXIR provides both the calming action of phenobarbitone (¼ gr.—16 mg.—per 5 cc.) and the tone-restoring effect of aneurine hydrochloride (5 mg. per 5 cc.).

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is plotted above the line. There is no relationship between the delay in the administration of Sulphapyridine and the duration of pyrexia within the limits of the time intervals under consideration.

(In cases marked X no Sulphapyridine was administered. The cases marked with a ? indicate those cases in which the time at which Sulphapyridine therapy commenced was uncertain.)

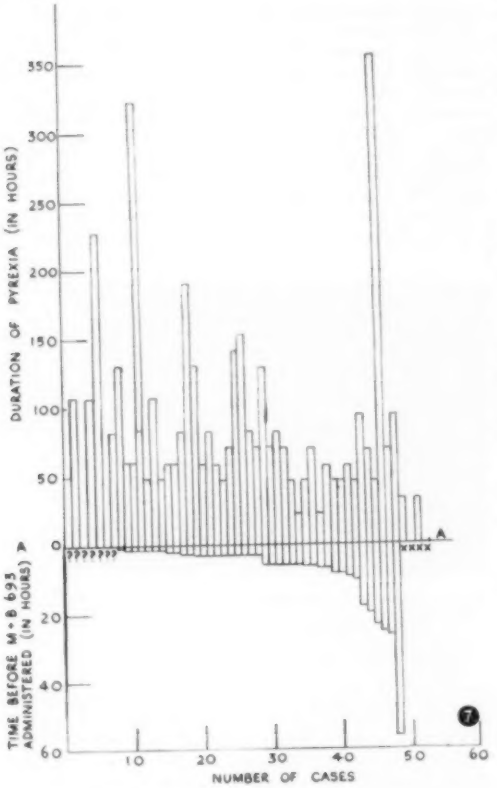


Fig. 7. Cases arranged in ascending order of periods before M & B. administered.

We are, therefore, in this series dealing with an acute disease which tended to receive treatment 22 hours after the commencement of symptoms and to receive chemotherapy, on an average, 7 hours after admission to hospital, and in which, after admission to hospital, pyrexia lasted for 72.96 ± 4.91 hours. The average stay in hospital was 5 days. After discharge from hospital, convalescence comprised light surface work and light underground probation; the former had a mean duration of 4.65 days, and the latter of 3.5 days. The mean total number of days lost from normal work was 13.15.

Recurrence. Of the 52 cases, 41 were still in the employ of the company 6 months after the original attack of pneumonia. Of these 41, 7 had had subsequent attacks of acute respiratory disease distributed as follows:

Right basal pneumonia, followed by right basal pneumonia after 3 weeks.

Right basal pneumonia, followed by right apical pneumonia after 5 months.

Right basal pneumonia, followed by left basal pneumonia after 4 months.

Right midlobe pneumonia, followed by bronchitis after 4 weeks.

Pneumonia, followed by bronchopneumonia after 3 weeks.

Bilateral, basal segmental pneumonia, followed by a similar infection after 3 weeks.

Bilateral, segmental pneumonia, followed by 6 pulmonary infections of various types; this case had bronchiectasis.

On examination 6 months after the original infection, 10 cases had signs in the chest, mostly rhonchi and crepitations, bearing little relationship to the site of the original infection in most cases. On further re-examination the signs had cleared in all cases.

Bronchograms done on cases with increased basal markings, and cases with recurrent attacks of acute respiratory infection, demonstrated one case of bronchiectasis.

Of 721 cases which recovered from pneumonia, 52, i.e. 7%, recurred. Of these, 5 had a second recurrence, i.e., 0.7% of the total. Of the 52 cases, the time interval between discharge and re-admission was obtained in 48 cases; of these 48, 19 recurred within the first 30 days of discharge from hospital, i.e. 3% of the original 721 cases.

Maynard [13] in 1913 found that of 1,129 Witwatersrand Native Labour Association Hospital 'Tropical' Natives who recovered from pneumonia, 80 developed a second attack within 30 days of the temperature's reaching normal, i.e. 7% of the 1,129 who recovered from the first attack. The distribution of the cases is shown below:

No. of Days	*W.N.L.A. 1913 No. of Cases Recurred*	*City Deep 1948 No. of Cases Recurred*
0	1	0
1	4	1
2	12	0
3	4	0
4	5	0
5	4	2
6-10	17	7
11-15	14	5
16-20	12	0
21-30	7	4

The recurrence rate is not high. The highest incidence of second attacks tends to occur shortly after the first attack and there is a tendency for second attacks to become less common as the interval after the first increases. The recurrence rate in the first 30 days has fallen from 70 per 1,000 in 1913 to 30 per 1,000 in 1948.

Complications. Although all manner of complications are encountered, they are rare, and usually of the mildest character.

Mortality. During 1948 there were 730 cases of pneumonia of all forms with 9 deaths.

The mortality in pneumonia for the industry as a whole has become more or less stabilized at a level of 0.6 per 1,000 per annum for all Natives employed.[15] The mortality rate from pneumonia before 1938 was generally more than 3 per 1,000 per annum.

(To be concluded)

JAW TUMOURS

III: ADAMANTINOMA

W. GIRDWOOD, CH.M. (W.W.RAND), F.R.C.S. (ENG.), F.R.C.S. (EDIN.)

Johannesburg

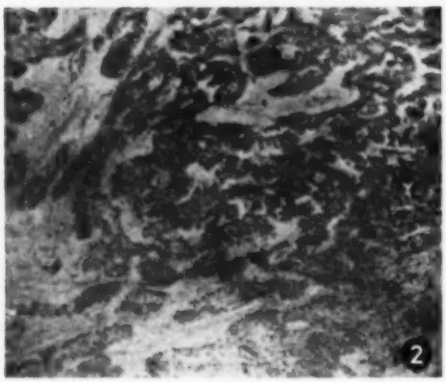
Adamantinoma is the common lower jaw simple tumour, presenting a cystic appearance on X-ray. These tumours are common in the Bantu[19] and when seen by the surgeon are usually large. The problems to be considered are:

1. Is an apparently benign cyst really an adamantinoma?
2. What is the histological picture (see Thoma) and is this a benign cystic lesion, or cystic and solid, or entirely solid?
3. What is the site and the nature of the tumour? This may influence the surgical treatment.

It is a good rule in the case of jaw tumours to suspect the obvious, as a cystic-looking tumour on X-ray may give all the features of one type of lesion and be found later to be an entirely different type of lesion. In all cases histological examination of linings of cysts and tumours is necessary. Histologically there is the variation such as Thoma describes.

The nature of the tumour is of interest[11] (Fig. 1). There are epithelial elements and a stroma. The epithelial elements are arranged in the simplest form as alveolar structures with high palisade epithelium surrounding the edge. There is usually a small or a large collection of fluid inside the alveoli with distension of the alveolar structure, so that on occasions one can only recognize a cyst with thinned-out lining or between the cysts a considerable amount of fibrosis. However, their alveolar structures contain a stellate reticulum when not over-compressed by the cystic distension. The stroma in the cystic types of lesion is not particularly cellular.

In some cases there is marked epithelial hyperplasia and papilliferous epithelial growths may extend into the cysts (Fig. 2) and there may be areas of solid white growth distributed amongst the cysts and their surrounding fibrous tissue. These cases of epithelial proliferation with solid growth occur especially in the upper jaw. On histological section there are masses of epithelial cells arranged as solid or papilliferous masses.



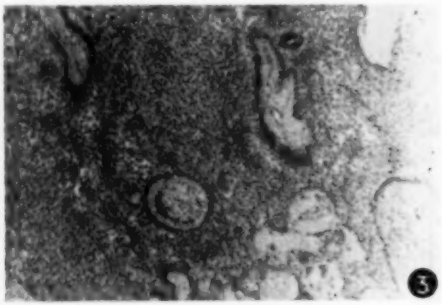


Fig. 1. Typical ameloblast layer and stellate reticulum in cystic adamantinoma.
Fig. 2. Adeno-adamantoblastoma.
Fig. 3. Apparently 'malignant' adamantoblastoma.

Cysts are still present and here and there the general arrangement similar to the cystic form may be distinguished. The stroma is usually like the cystic types, not particularly cellular. This type of lesion one could call the 'carcinomatous' or intracystic papilliferous or solid types. Gross structures and histology cannot be regarded as an index of rate of growth. There is a possibility of recurrence or malignant change in the adamantinoma.[9]

In other cases the stroma takes on an excessively active form (Fig. 3) and may have all the features of an active malignant tumour.[23] Metastases in the lungs,[22, 23] and lymph stands[22] have been described. These variations in the tumour may be associated with its tendency to local recurrence and local destruction although this is disputed.[9]

It is probable that the tumour is a type of basal cell tumour in its behaviour, arising as it does from the ameloblasts. These tumours cannot be treated medically. Cure can be offered only if complete removal is done. They are not encapsulated. In the regions of soft tissue false capsules are formed on the outside as layer by layer is destroyed from the inside. In relation to bone, the tumour is usually solid or cystic, without a capsule and without any definite evidence of its extent. Small cysts are often found deeper in the bone after one is fairly sure that the whole growth has been removed.

In a series of 379 cases extracted from the literature, Robinson[18] gives the following figures:—

Total number of cases: 379.
Sex in 311 cases:
Males, 45.7%.
Females, 34.3%.
Average age at the time of reporting: 37.6 years in 248 cases.
Average duration of the tumour: 8.5 years in 232 cases.
Average age at the time of discovery: 30.1 years.
Site of the growth in 293 cases:
Mandible, 83.7% in 247 cases.
Maxilla, 16.3%.
Structural characteristics in 219 cases:
Cystic, 57.5%.
Cystic and solid, 24.2%.
Solid, 19.1%.
Uncommonly, these tumours contain unerupted teeth and enamel, are pearly and show keratinization.
Malignancy or histological evidence of malignancy: 4.5%.

TREATMENT OF ADAMANTINOMA

The division of cases into the following types is made from the point of view of treatment:—

1. Peripheral alveolar types.
2. Central adamantinoma with thinning of the mandible as a rim, of the horizontal ramus.
3. Adamantinoma extending across the mid-line at the symphysis.
4. Adamantinoma of the ascending ramus.
5. Massive adamantinoma.
6. Adamantinoma of the upper jaw.

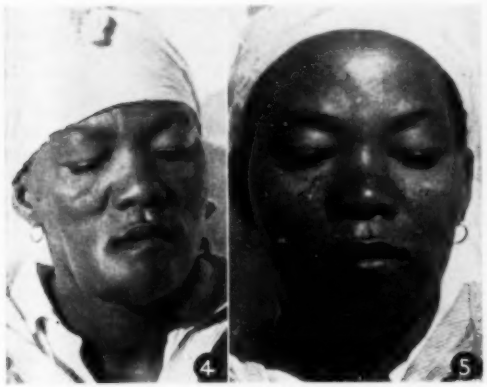


Fig. 4. Adamantinoma before operation. Peripheral cystic.
Fig. 5. Adamantinoma after operation.

1. *Peripheral Alveolar Types.* In these cases (Figs. 4-9) a cystic swelling occurs at the gum margin with expansion and destruction of the alveolar bone. The occurrence in the lower jaw especially in the Bantu, makes the possibility of adamantinoma very high. There is usually the typical trabeculated structure on X-ray and the cysts may be small or large.

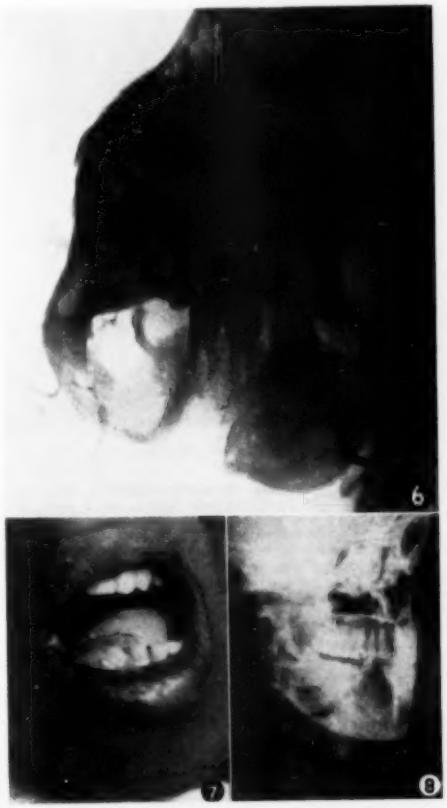


Fig. 6. Peripheral cystic. X-ray of adamantinoma.
Fig. 7. Adamantinoma. Peripheral cystic.
Fig. 8. X-rays of adamantinoma.

These tumours can be dealt with perfectly well by the method of biopsy excision, whereby the whole tumour is excised and the portion of the tumour in relation to the bone is removed as a whole, if possible, with a margin of normal bone around it. This is not always possible; then curettage of the tumour from the bone and careful gouging of the bone left is done, with nibbling away of the bone in relation to the tumour for the distance of about 1 cm. to remove any small extensions beyond the obvious regions of tumour growth. After this the cavity is cauterized chemically with carbolic acid and alcohol and diathermy coagulation if available. The mucosal borders can usually be closed over the remaining bone and drainage is effected for 48 hours.

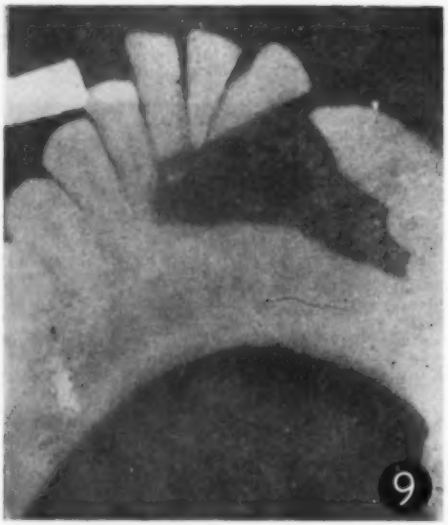


Fig. 9. X-rays of adamantinoma.

Case History. Gasemeru, a female, aged 36 years, had a swelling of the right lower incisor region for 8 months. It had grown slowly and painlessly and all the teeth were still in position. On examination there was a swelling of the mandible in the right lower incisor region with visible expansion of the bone into the mouth, interior to the teeth on the right side. The tumour felt cystic intra-orally but solid externally. An X-ray showed erosion of the alveolar portion of the bone in the region of the left incisor tooth. There was absence of the teeth on the right side, from the first molar to the lateral incisor. There was evidence of trabecular expansion of the tumour in the right incisor region with a projecting soft tissue swelling.

5 July 1944. Excision of tumour intra-orally with curettage and cautery was done.

21 December 1948. No recurrence was found.

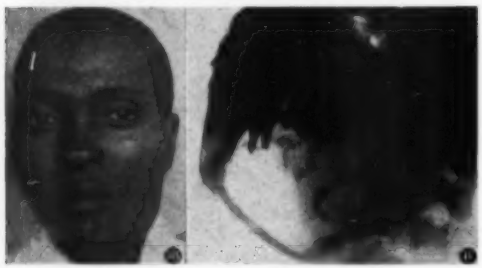


Fig. 10. Adamantinoma.
Fig. 11. Adamantinoma causing shell of mandible. Rim of bone remaining.

2. *Central Adamantinoma with Thinning of the Mandible as a Rim of the Horizontal Ramus.* In these cases (Figs. 10 and 11) it is undoubtedly true that if the rim is not removed and a formal excision is not done, the chance of recurrence is considerable. Resection of the tumour, by resecting the jaw above and below the involved portions of the mandible, gives the best hope of a cure (Ivy).

The alternative method is to turn mucosal flaps down, remove the tumour and curette the cavity. The body walls of the cavity are nibbled away until there is a flat surface of bone remaining apparently unaffected and the mucosa is then closed over it. In any case, if the bone is thinned it is wise to support the mandible by extra-oral splintage; or, if fracture occurs, to have available metal cap splints or wiring to maintain the normal position of occlusion of the teeth. There are cases where, because of the condition of the patient, the less severe operation is sometimes done; but in the case of the ramus of the mandible from the angle and interior ramus, excision is not a difficult procedure. In the good risk case it is advisable and reconstruction by bone graft can be done.

Inadequacy of primary curettage in early cases is demonstrated by the high percentage of recurrences after primary conservative treatment (79% recurred—Kegel). Curettage followed by chemical and thermal cautery was advocated by Bloodgood in monocystic or solid lesions with an intact bone shell. The tumour is removed, a carbolic acid swab followed by alcohol is applied and then a 50% solution of zinc chloride; thermal cautery follows. The expanded bone shell is resected subperiosteally (Kegal).

This procedure will cure an adamantinomatous dentigerous cyst, giant cell tumour or central fibroma. Simple curettage cures only dentigerous and root cysts.

3. *Adamantinoma Extending Across the Midline at the Symphysis.* In these cases (Figs. 12-16) excision of the mandible will leave serious and even dangerous complications:

1. The attachments of the hyoid and tongue muscles to the mandible are lost and the tongue may fall back and cause obstruction to breathing.

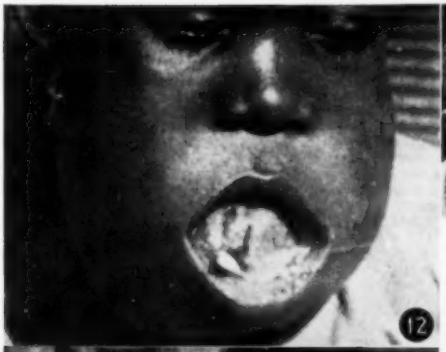
2. The two halves of the mandible will tend to fall together and occlusion will be lost.

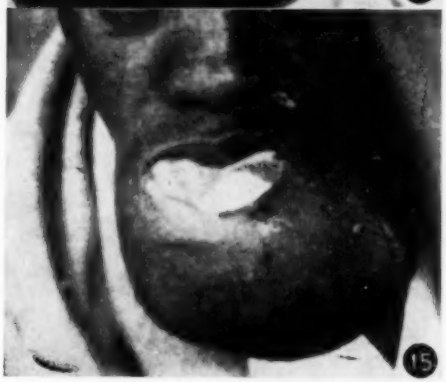
If the symphysial portion of the mandible is removed, it is important to maintain the rest of the jaw in proper position and, by suturing the hyoid forward, usually to the extra-oral apparatus, to ensure that the tongue does not fall back. A prosthetic apparatus can be fixed by metal cap splints to the remaining lower teeth and a form of bar and attached bun accommodated for buccal inlay grafts if necessary, either at the time of operation or later. This procedure is complicated and sometimes dangerous to life and, in some patients, cannot be done for reasons of the general condition present.

The alternative of excision of the tumour, nibbling away of surrounding bone, saucerizing the cavity and cauterization, may be done in poor risk cases without danger to life, but the possibility of recurrence remains, requiring further intra-oral treatment at a later date.

4. *Adamantinoma of the Ascending Ramus.* In these cases (Figs. 17 and 18) the swelling underlies the parotid externally and, intra-orally, exposure is complicated by the overlying internal pterygoid, the origon of the superior constrictor and the buccinator muscles, and other difficulties such as vascularity of the pterygoid region.

In these cases an extra or intra-oral exposure for local excision of the tumour is practically impossible. In such cases, a formal resection of the mandible with disarticulation at the temporomandibular joint is the treatment of choice and resection is done distal to the tumour.





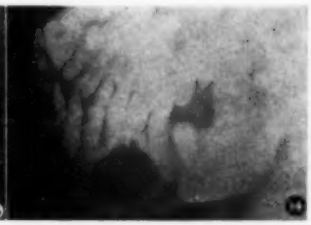
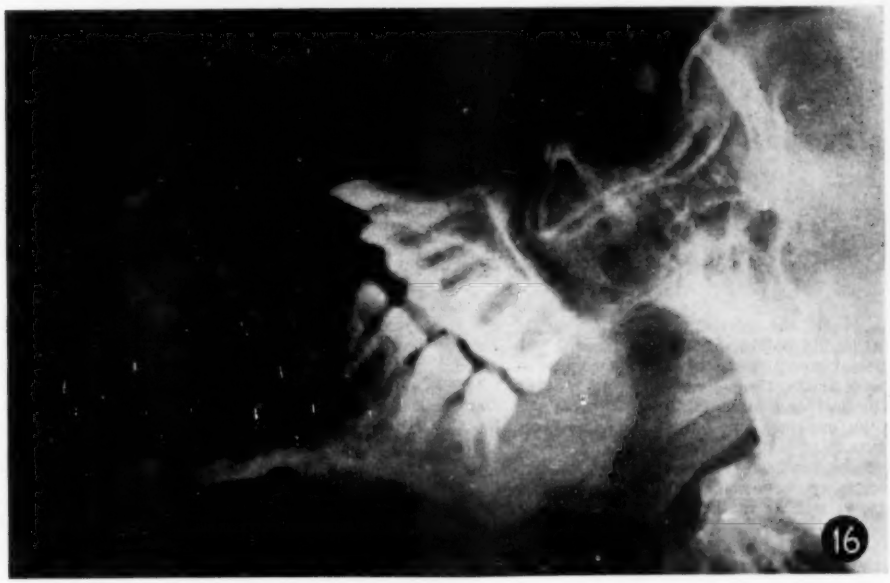


Fig. 12. Adamantinofibroma.
Fig. 13. Adamantinoma extending across the symphysis.

5. *Massive Adamantinoma.* It is not unusual in the Bantu for cases (Figs. 19 and 20) to be seen which have had the jaw tumour for up to 20 years. Franz and Stix [7] quote a case of presence of the tumour for 51 years. In fact, in all types of jaw tumours, it is the rule rather than the exception for tumours to present in the massive form. In these patients cure can still be offered by complete excision of the tumour and resection of the mandible distal to the growth.

Surgical Exposure. For excision of massive tumours of the jaw,[1] intra-oral or even classical external incisions are insufficient to obtain exposure, especially when the tumour has extended into the infratemporal fossa or is bulging the soft palate. Most cases with massive tumours were adamantinomata; but in some instances, by the use of this incision it was possible to operate on other tumours which would have been considered inoperable by the usual standards. The advantages of this approach are that



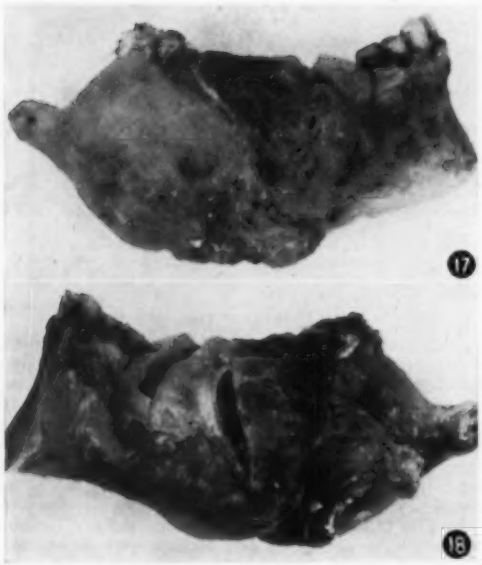
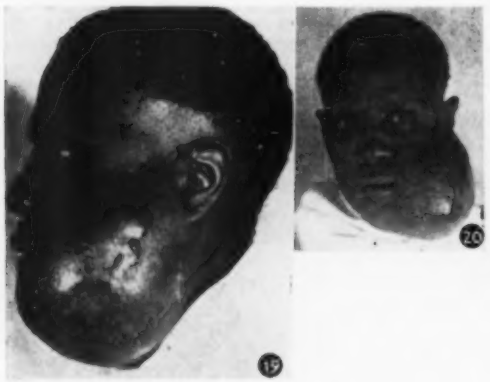


Fig. 18. Adamantinoma of the angle and ascending ramus.



an extension into the infratemporal fossa and pterygoid region can readily be visualized and removed. Maxillary or mandibular tumours can be approached through this incision and, finally, there is no danger of failure of the incision to heal and leave in the face, after maxillary excisions, an unsightly hole with the unpleasantness that this involves.

This incision has been employed in cases of carcinoma of the antrum and of a massive mixed parotid tumour of the palate, as well as in the massive cases of adamantinoma.

Case History. Michael Litale, Bantu male, aged about 50–60 years, gave a history of a swelling of the jaw for the last 17 years. The swelling followed extraction of teeth from the lower left side of the jaw. There was no tenderness or pain but the patient went to a doctor in 1927 when the tumour, according to the patient's statement, was excised. The tumour recurred and excisions were done in 1936 and 1941.

27 August 1943. The left side of the face over the mandibular region was occupied by a large tumour mass. The swelling extended from the ear to the midline on the left side. There was a scar at the apex of the mass which was adherent to the underlying swelling, which was hard in some parts but cystic in others. It felt hot but there was no definite tenderness. The cervical glands were not palpable. The condition of the mouth was unhealthy. The teeth were all present except the left lower premolars, molars and the canine. General examination failed to reveal any other abnormality. In the mouth there was a large cystic bulging of the mandible with some surface ulceration in the alveolar region on the left lower side.

Fig. 21. Massive adamantinoma.

An X-ray (Fig. 21) showed complete destruction of the left half of the mandible with erosion of bone extending to the region of the right canine. A soft tissue swelling could be seen outlining the lateral part of this swelling. There was coarse trabeculation throughout the tumour. No definite evidence of a left condyle could be seen.

The clinical diagnosis of adamantinoma was made by reason of the long history of a recurring tumour which had caused local destruction of the mandible and revealed trabeculation on

Treatment. It was decided to remove the tumour and to provide local pressure by means of a pressure bun applied to a prolongation bar. The bar was made to screw into position on cap splints. A training flange was also provided to prevent deviation of the jaw after operation. Upper cap splints were

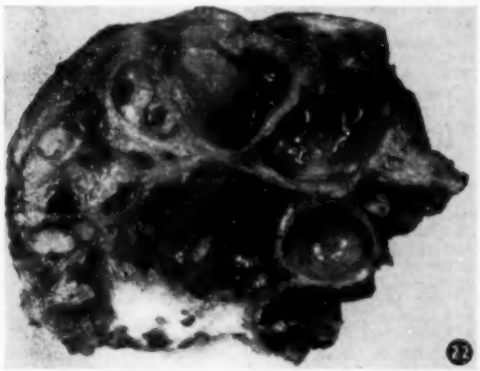
also made to lock the jaws if necessary. The usual blood transfusion of 1,000 c.c. was started just before operation and continued throughout it. The anaesthetic was intratracheal gas and oxygen with a good pack-off.

The incision was made on the outer aspect of the growth and surrounded the adherent scar, leaving it attached to the tumour. The incision was continued into the mouth and the facial flaps were elevated and raised from the tumour. In the case of the upper flap, the zygomatic arch, the infra-orbital vessels and the angular process were exposed. Resection of the zygomatic arch followed and the temporal muscle could be seen. The tumour was removed inside its false capsule. The false capsule consisted of the periosteum of the mandible below with the masseter muscle attached to it. By cutting through the masseter, removal of the mass was facilitated. The temporal muscle and temporal fascia in the upper part formed the false capsule and it was only inferiorly that one could not remove a definite layer of false capsule with the tumour. Below, the tumour was in direct contact with bone and shelled out. In this region the tumour was a white, homogeneous compact growth very much like a carcinoma; whereas in its upper part it consisted mostly of cystic spaces. The mandible in relation to its lower part was nibbled away to the level of the canine on the right side and the bone carbolized. An extensive hole was now left, with considerable oozing in the region of the infratemporal fossa and the pterygoid muscle stumps left after removal of the tumour.

A careful mould of Stent's composition was then taken of the raw area and fitted around the prolongation bar which had just previously been fixed by screws into the lower right cap splints. The mould and bar, upon cooling, were removed and a Thiersch graft applied around the mould. This was then fitted into the cavity and the prolongation bar screwed back into position. The large raw cavity was thus filled with a closely fitting mould held firmly in position and covered with a skin graft to the raw area. The training flange was then fitted onto the right lower cap splint and the cheek flaps were sutured by interrupted silk and a pressure dressing was applied. This dressing consisted of acriflavine emulsion cotton wool squeezed out and then bandaged on firmly, by means of a crêpe bandage over gauze and cotton wool.

The patient's general condition was good after operation and none of the complications expected, i.e. inhalation of blood, atelectasis, pneumonia or sepsis, occurred.

The Stent's mould was removed by chipping off pieces with bone-nibbling forceps and then, when small enough, extracting the rest. The mould was left *in situ* for 3 weeks before extraction. After removal the cavity was painted with mercurochrome. Very little raw area remained and the side of the face was natural in appearance and did not fall in to any great extent. Salivation was troublesome for some weeks but this passed off slowly.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No plastic surgery excision of scars was considered necessary and the patient removed the cap splints and flange after 2 months. He was followed up for 2 years without any evidence of recurrence. There were no palpable cervical glands and the patient was completely satisfied with his ability to chew. There was some swing over to the side of the jaw removed, but his general appearance was not unsightly and he obtained work as a lift boy, having been unemployed for the previous 7 years.

Michael reported again in March 1946 with a small swelling in the region of the symphysis of the mandible, obviously a recurrence but still small and amenable to treatment. He was advised to enter hospital for further surgical treatment but he disappeared.

In February 1949 he had the symphyseal recurrence excised with carbolization of the bone related to the growth. Progress was satisfactory.

The specimen was typical of adamantinoma (Fig. 22). There were large cystic spaces which contained brownish watery fluid, sometimes mucoid material. The walls of these cysts were thick and fibrous with gritty bone or calcification. There were areas of epithelial papilliferous projections into the cysts. In some areas there was a solid epithelial mass of tissue somewhat resembling carcinoma, but in others there was looser tissue, like masses of papilliferous processes. The histological sections showed cystic space with the typical palisade epithelium. The stroma particularly seemed to be the most cellular element in the growth.

Discussion. There was no droop of the side of the mouth in spite of the cutting of the facial nerve to the lower lip (Fig. 23). The buccinator muscle compensated for this.


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Inside the mouth the skin graft became almost black, much darker than the surrounding mucosa, but it softened to perform an efficient protective function without ulceration.

The recurrence was not unexpected in the region of the symphysis and treatment for this was not difficult. In spite of its very cellular nature, no metastases were present outside the local site of the growth.

With regard to massive growths, whether adamantinoma, carcinoma or other neoplasm of the mandible or maxilla, it was thought that, if the zygomatic arch could be elevated or resected, an exposure of the infratemporal fossa could easily be effected. Once this was done the temporal muscle, the ascending ramus of the mandible and the pterygoid muscles could be visualized and adequate resections done under vision.

The incision therefore for both mandibular and maxillary tumours started from the angle of the mouth, down to the line of the mandible and then posteriorly to a finger's breadth below the lower border of the ear. The muscles of the lower lip were cut with scissors and the whole flap of the cheek was elevated by cutting along the inferior buccal sulcus. The buccinator muscle was cut from its attachments to the mandible below and from the maxilla above. The whole facial flap was thus turned upwards and outwards, containing the buccinator, masseter, the facial nerve and the parotid. The only branch of the facial nerve cut was that to the lower lip, but the buccinator was still present with its nerve supply and the cervical branch to the lower lip was still retained.

The upper part of the wound was deepened down to the maxilla and the zygomatic arch, and the flap elevated above these. The arch was then resected. The lower part of the incision could be adjusted according to the size of the tumour.

Massive tumours of the mandible and maxilla can be removed through this type of incision without any really serious deformity. The incision always heals without ever any danger of the hideous deformity sometimes obtained when the usual incision for excision of the maxilla breaks down, as it sometimes does, leaving a deep hole in the face which reveals the tongue, nasopharynx and the base of the skull, while the discharges from these areas are uncontrollable and run unpleasantly over the remains of the face.

In all cases pressure moulds were applied to raw cavities and raw areas and skin grafts were applied at the time of the operation. The pressure pads were either moulds of Stent's composition or of acriflavine wool pressure pads sewn in or adapted by special dental splints. The advantages of pressure pads and skin grafting are that there is no post-operative bleeding and inhalation of blood, the greatest danger in these operations. The raw areas heal rapidly so that a chronic septic cavity and the danger of inhalation pneumonia, or other septic complications are avoided; also, the mould helps to keep the tissues in their normal positions so that they become adapted to them and configuration is maintained.

It is always an advantage to have dental splints applied so that, by means of a flange, the jaws can be prevented from swinging out of occlusion and a prosthesis can be applied to maintain the skin of the face in position after removal of the bony structures; also to fit a palate prosthesis to assist speech at a later date. All these considerations must be remembered whenever a jaw tumour is under treatment. In the upper jaw, bone grafting has no place as the cheek can be held up by a prosthesis; but in the lower jaw, although many Natives will be perfectly happy to eat with one half of the jaw and will often discard complicated prostheses, bone grafting is indicated to give a platform for dentures and to fill out the concavity following the excision of one side of the mandible.

6. *Adamantinoma of the Upper Jaw.* These are usually solid and behave in a manner indistinguishable from carcinoma of the antrum.[2, 4]

Case History. T. Ngabunde, a female Zulu about 60 years old, was admitted on 12 May 1945. Seven months before the patient noticed a swelling in the roof of the mouth. Pain started 3 months ago. The pain was related to the tumour and the whole of the right side of the face. The left nostril had been blocked for 3 months with bleeding and the left eye had 'watered' for 3 months.

On examination there was a mass related to the left maxilla. The left eye was slightly raised and there was bulging of the hard and soft palates. The uvula was displaced to the right. The tumour felt rubbery, without ulceration and was the size of a small orange. The skin of the face was attached to the underlying tumour. The molar and premolar teeth were missing in relation to the tumour and there were numerous loose teeth with dental sepsis.

At operation a solid epithelial growth was found to extend into the base of the skull so that only palliative excision was possible. Histologically it was a solid adamantinoma.

SUMMARY

1. Adamantinoma must be considered in the differential diagnosis of all jaw tumours, especially in the Bantu. Many conditions appear to be one thing and turn out to be another.

2. Histological types of adamantinoma are numerous.

3. Cystic, mixed and solid forms occur. The upper jaw adamantinoma is usually solid and behaves as a malignant tumour.

4. The clinical types of adamantinoma can be divided into:—

(a) Peripheral or alveolar type.
(b) Central adamantinoma with a thin rim of the horizontal ramus.
(c) Symphysial adamantinoma.
(d) Adamantinoma of the ascending ramus.
(e) Massive adamantinoma.
(f) Upper jaw adamantinoma.

5. The principles of treatment vary according to the clinical types and reconstruction must go hand in hand with excision.

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VERENIGINGSNUUS : ASSOCIATION NEWS

NATAL COASTAL BRANCH

THE ECONOMIC CRISIS IN MEDICINE *

DR. A. BROOMBERG
Durban

In casting around for a theme for the address which is customarily expected from the President on the expiry of his year of office, I came across an address delivered over half a century ago by the then Dr. William Osler. Speaking in Troy, New York, on the occasion of the semi-centenary of the local hospital he chose as his subject *On the Influence of a Hospital upon the Medical Profession of a Community*, and concluded a wise and eloquent oration with the following words: 'Fortunately the medical profession can never be given over wholly to commercialism, and perhaps this work of which we do so much and for which we get so little—often not even thanks—is the best leaven against its corroding influence. While doctors continue to practise medicine with their hearts as well as with their heads, so long will there be a heavy balance in their favour in the bank of Heaven—not a balance against which we can draw for bread and butter, or taxes or house rent, but without which we should feel very poor indeed.'

And although Osler's thoughts were turned towards those who were doing an enormous amount of *pro deo* work in hospitals, work which he felt was worthy of some sort of remuneration, I could not help associating the idea of the balance in the 'Bank of Heaven' with the present-day economic circumstances and their influence on the profession. I do not think that anybody will deny that some commercialism has crept into the ranks of practising doctors and that because of this, the profession has been subjected to hostile criticism by the press, the public, and particularly by aspiring candidates for political honours who have always found the slogan 'Enslave the doctors, and provide a free medical service'—most productive from the point of view of vote-catching. But at the same time I make bold to say that the vast bulk of doctors practise their profession in the spirit of Hippocrates and that the only big balance which they do succeed in building up is the one in the Bank of Heaven. And so, however one may deplore the actions of the unscrupulous who have allowed themselves to become brutalized by the rapacious competitive economic system under which we live, and who have battened and fattened on illness and human suffering, one is consoled by the knowledge that these 'black sheep' constitute so small a fraction of the medical body corporate as to be dismissed with the contempt which they deserve.

There is, however, another aspect of practice which to my mind is of much greater importance. It is the question of economic survival. In season and out the cry has been raised by the Press and the public that the cost of doctoring has become so high that only the very wealthy and the very poor can afford to indulge in the luxury of illness. We ourselves have at times commented on the apparently increased cost of medical care to the people. But we must not forget that the tremendous advances in modern methods of diagnosis and treatment have in some measure themselves been responsible for these increased all-round costs. The modern hospital will still continue to provide the poor with all that is best in medical care. It does that unstintingly. No man need be deprived of the best which medicine can give if he feels disposed to avail himself of the facilities of a modern general hospital. In private practice, however, where the doctor has to provide his own equipment, his own drugs and instruments, and furnish his rooms in a manner adequate to deal with his work and the convenience of his patients, initial capital costs and subsequent maintenance can be very heavy indeed, and the fees which he charges for his services must necessarily bear some economic relationship to these factors. Not only must he recoup himself for his capital outlay, but he must provide for maintenance of the service and also for the subsistence of himself and his family in the present and as far as possible make some provision for the future. Now all this will appear to be platitudinous. It is self-evident, common-sense economics, and it is equally self-evident that when costs go up something has to be done to meet those rising costs. Business men know this only too well. They simply raise the prices of consumer goods, and as costs go higher and ever higher, and as wages increase and as maintenance soars to higher and higher levels, so business men and property owners elevate their prices and their rents to maintain themselves and the services which they provide. Who of us has not had the experience of feeling robbed by the garage man, the refrigerator repairer, the plumber, the taxi driver and every other artisan who has ever been called in to do a job of work for us. We have groaned at the increased rentals we have been called on to pay, at the salaries we have to pay our technicians, our typists, receptionists, and what have you. We have all listened with painful expressions to the tales which our wives tell us, of the fantastic increase in the cost of food, of clothes, of all the essential things of life. We pay for the motor-car on which we depend for our livelihood 3 and 4 times as much as what we did 10 years ago, and above all else we pay back in taxes fully a quarter of all that we earn. Yet with all this we try to maintain a certain standard of living, a certain minimal social status in the society in which we live.

The cost of living has jumped over 300% and with the exception of the doctor every other profession, trade, industry and business has raised the price for its services or its products. The public has not uncomplainingly paid these exorbitant prices. Not without complaint, but certainly not with the same outraged feelings aroused by the presentation of the alleged excess of the doctor's bill, a bill rendered for services honestly, conscientiously and honourably performed in the great work of relief of human pain and the alleviation of human suffering. It is perhaps unfortunate that the doctor, like every other living being, must eat, clothe himself, house himself and provide himself with the amenities of life, and it is still more unfortunate that in common with his fellow men he must ensure that he earns the customary means of exchange to enable him to do so. He just has to purchase his needs with money, and so he has to charge a fee for his services. The Bank in Heaven will not provide for these. All that his balance there will provide is a problematical front seat in Paradise and more often than not a charitable grant from some benevolent fund for his dependants in the event of his arriving at that desirable destination.

Now the basis of remuneration for a service depends on very many factors not the least important being the degree of training required for the most efficient performance of that service and its indispensable necessity in the interests and well-being of a community. In considering these two factors only and excluding numerous others, it will be generally agreed that the training of the modern doctor is as rigorous and exacting as that of any other profession and more so than most; that it is the most costly to learn and to practise both in terms of money and endurance; and that above all else it is absolutely indispensable in the promotion of the health and general welfare of any community whether it be civilized or primitive. The health of the individual is paramount for the welfare of society. The doctor's task is to maintain and promote that health by every means in his power, and for that work he is entitled to ask society to remunerate him on a basis commensurate with the rigor and costliness of his training, the arduousness and responsibility of his duties, and above all, the contribution which he makes to the sum total of human health, welfare and the stability of the society whose great and solemn task is his to protect from the ravages of disease, suffering and all the distress, both personal and communal, attendant thereon.

* Presidential Address delivered at the Annual General Meeting of the Natal Coastal Branch on 13 February 1952.

The service rendered by those who practise medicine cannot be assessed in terms of money in the same way as the buying and selling of goods. There is that indefinable humanitarian spirit behind it, so clearly expressed in the ancient oath of Hippocrates and thousands of years later re-affirmed in the Declaration of Geneva, which lays it down that doctors shall give their all to those who seek their aid. They shall practise from their hearts, and not because of any anticipated material profit which may accrue as a result of that giving. That is perhaps the reason or one of the reasons why the doctor's fees have remained comparatively unchanged in spite of the most revolutionary economic changes which the present century has yet experienced.

Whilst I am firmly convinced that the time has arrived for the profession as a whole to give the most serious consideration to its economic position, I wish here to refer more specifically to that section which does not habitually command the highest scale of remuneration for its services. I wish to deal more particularly with the general practitioner. In 25 years he has, almost shamefacedly and apologetically, tried to meet a 300% rise in his cost of living by raising his fees 30%. As against this increase, however, his share of *pro deo* work has not become any smaller. His bad debts have increased. The growth of free hospitalization has not only cut down the field of private practice, but has at the same time demanded from the doctor more and more unpaid or underpaid service; but by far the most significant development has been the growth of Medical Benefit Societies and Medical Aid Societies. In the former, payment is made to a closed panel of doctors on a capitation or sessional basis; in the latter, payment is made to an open panel on the basis of a preferential tariff of fees, the tariff representing a reduction of at least 30% of the fees normally charged in private practice. The effect of the Benefit Society is to divert large groups of potential patients from the private practitioner to the limited panel, usually of part-time men who have to work very hard for very little, e.g. The Railway Sick Fund. The effect of the Medical Aid Society is that fees have been pegged down to a pre-inflation level and are kept at this level by reason of the agreed tariff. On the one hand thousands of potential fee-paying patients are prevented from exercising the right of free choice of doctor, while on the other those who are members of Medical Aid Societies can avail themselves of the services of the profession, but at a rate of remuneration which to the doctor is, under present conditions, grossly uneconomic. Let us not forget that over 300,000 people in this country are catered for by one or other type of contract practice organization. Nor must we forget that with the growth of industry, these Sick Funds and Medical Aid Societies are growing in number from day to day, so that the field of private practice is becoming more and more restricted. From the point of view of the working and salaried man there is, of course, a tremendous advantage in being insured against the payment of formidable medical expenses and I have no quarrel with such insurance. I am prepared to agree also with the advantages to the medical practitioner in that he incurs no bad debts in his dealings with members of Medical Aid Societies, since by the nature of their constitutions they undertake to pay accounts in full on the basis of a preferential tariff, but I am very concerned with what appears to me to be the inevitable outcome of the contract-practice system.

Whilst originally Medical Aid Societies were formed to assist employees of a specific trade, industry or commercial group and membership was confined to those employed in these groups, the position in recent years is that non-specific societies have been and are being formed to cater for any person who wishes to join, whatever his occupation and, within certain limits, irrespective of his income. These societies are actually canvassing for members on a national scale and offering to their members the benefits of the preferential tariff.

The Government of the country is also considering a similar type of nation-wide Medical Society and will quite possibly present a Bill on those lines to Parliament in the present session. We face the peril, therefore, of being ground down between two enormous millstones. On the one hand, the Benefit Funds to which very large numbers of industrial employees are now compelled by law or conditions of service to subscribe, and on the other an ever increasing circle of Medical Aid Organizations. I cannot over-emphasize what all this will eventually mean to the profession in terms of economics; the constriction of the field of private practice and free choice of doctor is inherent in the Medical Benefit Fund system; virtual pegging down of fees in the other. I do not know of any other profession which finds itself in a similar dilemma, nor do I know of any other occupation (industrial or trading) which has entered into or allowed itself to be forced into an iron-clad arrangement whereby its fees or earnings or profits if you like, are kept down at a level which manifestly bears no realistic relation to the stark fact of the rise in the cost of living. The situation is quite Gilbertian, for we as a profession have done this voluntarily and deliberately with no thought to the future. The specialist group as well as the general practitioner is vitally affected, particularly the latter, for the G.P.'s fees are so much smaller even though his range of practice may be more extensive. For him it is an exceedingly serious matter if for any reason he is no longer accessible to the public or the public to him, or if for any reason his fee-earning potential becomes restricted. It is no secret that in addition to the factors which I have already mentioned, there are certain fields of medicine from which he is gradually being ousted by virtue of the situation whereby the public has free access to the specialist. That is a matter which I do not wish to pursue any further here. I mentioned it merely to emphasize the precarious economic state of general practice when considered from various aspects.

The facts as I see them are that the doctor's services are in terms of money far less costly to the public to-day than those rendered by any artisan. Do I exaggerate when I maintain that the average minor illness costs the man in the street a great deal less than having his refrigerator repaired, or his car overhauled or having a couple of feet of water piping replaced by the plumber. The G.P.'s visiting fee is less than that of the piano tuner. It will cost John Doe very much less to engage medical care for his peptic ulcer than to divorce his wife, but then he does not divorce his wife every day. The taxi driver's fare for a distance of 4 miles is higher than the medical practitioner's fee if the latter is called to attend a Medical Aid Society patient, and the taxi driver is not normally called upon to examine his fare, however charming she may be, nor has he to assume any responsibilities for the well-being of that fare from the medical point of view. I quote these few examples to bring forcibly to your notice the gross inadequacy of the remuneration which the average doctor receives in these days of economic crisis. It is indeed obvious that his high status in society he occupies not by reason of his earning potential but by reason of his noble vocation. His fees in fact bear very little relation to reality or to the vast importance and the indispensability of the service which he renders to his fellow men.

The time has come for us to stop fooling ourselves, to stop being made the objects of exploitation by others. We have been asked to give and concede. We have given and we have conceded. We have for generations given of our time, our ability and our service *pro deo* to the poor and the needy, both in private practice and in hospital practice. We have for years given our all in Government service at rates of pay which no self-respecting motor mechanic would look at. We have willingly given preferential tariffs to Medical Aid Societies, deliberately placing a halter around our necks, and I have no doubt that further and further demands will be made until the day comes when income and expense will no longer be able to balance and the younger members of the profession will find nothing but starvation facing them. The red light is showing and the time is long overdue for stock to be taken and for a remedy to be sought. A halt must be called and a remedy sought for this all but inevitable economic strangulation with which the profession is faced. The alternative might well be the degeneration of medical practice into a trade brutalized by competition and bargaining —in other words—commercialism. The signs and symptoms of this degeneration are already quite plainly evident. But with all that, with all the cogency for the argument for raising our remuneration to a level commensurate with the present-day cost of survival, I am satisfied that for the men and women who practise the art of healing in the spirit of that art, there will always be a Balance in the Bank of Heaven, for the guiding light of pity, sympathy and selfless devotion will ever illumine the path of those who have devoted their lives to the ceaseless battle with pain, disease, suffering and death. Even though we cannot draw on that balance for our bread

and butter or for our taxes or for any of those material things which are necessary for our health and survival, even though we must face the stark realities of the facts and figures as they appear in the Earthly balance sheet, in spite of all these things, the physician will still do the work, and if I might paraphrase the words of the great Osler: 'He will carry on his task as he was meant to do. He will still be the adviser and the valued friend in every household into which he is brought. Few men live lives of more devoted self-sacrifice than he does, but there is the danger in this treadmill life that he lose more than health and time and rest—his intellectual independence. Even in populous districts the practice of medicine is a lonely road which winds uphill all the way and a man may easily go astray and never reach the Delectable mountains unless he early finds those shepherd guides of which Bunyan tells: Knowledge, Experience, Watchful, and Sincere. The circumstances of life will mould him into a masterful self-confident, self-centred man, whose worst faults often partake of his best qualities. The peril is that should he cease to think for himself he becomes a mere automaton, doing a penny in the slot business which places him on a level with the chemist's clerk, who can hand out specifics for every ill from the pip to the "pox".' It is not only intellectual but also economic independence that he must strive to preserve at all costs, for the former hinges so much on the latter. Not to bargain, not to become the slave of any system but to give willingly the best of which he is capable, in return for a remuneration commensurate with his abilities and the contribution which he makes to the health, happiness and social stability of his community.

TAK ORANJE-VRYSTAAT EN BASOETOLAND

NOTULE VAN DIE JAARVERGADERING GEHOU IN DIE BLOEMFONTEIN-KLUB OP SATERDAG, 23 FEBRUARIE 1952

Aanwesig: Ses-en-dertig lede, van wie 7 van buite Bloemfontein. Dr. J. N. W. Loubser, die President, het voorgesit en die aanwesiges verwelkom.

Presentasie van Sertifikaat van Emeritus Lidmaatskap aan dr. S. M. de Kock: Die President het dr. Raymund Theron, President van die Mediese Vereniging van Suid-Afrika, gevra om die presentasie te doen. Dr. Theron het gewag gemaak van al die dienste wat dr. de Kock oor lange jare aan die beroep en die Vereniging gedoen het.

In antwoord het dr. de Kock sy dank uitgespreek vir die eer en die Sertifikaat. Doofheid het dit vir hom onmoontlik gemaak om deel te neem aan die verrigtinge van die Vereniging en bydraes te maak waarvoor hy die tyd en krag het.

Toe hy in Bloemfontein begin het was die blanke bevolking 10,000 en daar was slegs 5 dokters. Sy eerste rondtes het hy te voet gedoen. Agtereenvolgens het hy 'n trapfiets, 'n wit perd, 'n kar en perd, 'n motorfiets en 'n motorkar aangeskaf. Die vroeë jare was gelukkig. Die dokters was almal algemene praktisyns. Wat die toekoms betref voel hy dat daar twee probleme is wat aangepak moet word as die agting en status van geneeshere bewaar moet bly:

1. *Die status van die algemene praktisyn.* Spreker voel dat die publiek te veel aan die spesialiteite vir hul dienste moet betaal en te min aan die algemene praktisyns. Hy meen ook dat spesialiteite meer as konsultante moet optree.

2. *Die kleurskeidslyn.* Medici moet die saak aanpak. Spreker voel dat ons plig duidelik is en dat ons moed moet hê. Die Hippokratiese eed ken geen skeidslyn gegrond op geslag, geloof of ras nie. Die probleem moet taktvol aangepak word. Indien daar verskil van sienswyse is moet die medici saam staan, die minderheid moet hom die wil van die meerderheid laat welgeval en nie afstig nie.

Hierna het dr. de Kock die vergadering verlaat.

Verkiesing van Ampsdraers: Dr. Loubser het nou die President se insignia aan dr. A. J. Groenewald, *die nuwe President*, oorhandig en hom gevra om die stoel te neem.

Onder-President: Drr. Hesselberg en Connan is voorgestel maar wou nie staan nie.

Dr. C. H. Derksen is deur drr. P. Connan en J. N. W. Loubser voorgestel en is eenparig gekies.

Ere-Sekretaris: Dr. Beck de Villiers is gekies.

Ere-Tesourier: Dr. J. G. Muller is gekies.

Lede van Takraad: Die volgende is voorgestel: Drr. M. I. Goddefroy, E. Hesselberg, P. Connan, A. L. Ferguson, J. W. van der Riet, P. J. P. van Blerk, J. W. Wessels, R. S. Verster en van Coller.

Drr. Ferguson, Verster en van Coller was nie bereid om te staan nie.

Na stemming per stembrief is die volgende gekose verklaar: Drr. P. Connan, P. J. P. van Blerk, E. Hesselberg en J. W. van der Riet.

Verslag van Ere-Sekretaris: Die Ere-Sekretaris het sy verslag voorgelees.

Verslag van Ere-Tesourier: Die Ere-Tesourier het met sy verslag gecouditeerde state voorgelê. Besluit is om 'n bedrag van £73 1s. 6d. aan uitstaande ledegelde af te skryf as onverhaalbaar.

Dr. C. D. Brink het 'n beroep gedoen dat uit die surplus 'n projektor gekoop word en 'n bydrae tot die Liefdadigheidsfonds gemaak word.

Dr. J. S. Visser het voorgestel dat geld gebruik word vir die aankoop van joernale.

Dr. C. H. Derksen het 'n teenvoorstel gemaak dat geen geld aan boeke uitgegee word nie maar dat die surplus in 'n reserwefonds bly vir 'n moontlike strydfonds in die toekoms.

By stemming is Dr. Derksen se voorstel aangeneem.

Verslae: *Afdelings*:—

1. *Noordoostelike*: Dr. J. N. W. Loubser het hierdie verslag voorgelees.

2. *Sentrale*: Dr. B. L. Cockcroft het verslag gedoen.

3. *Noordelike*: Dr. C. F. G. Troskie het verskoning gemaak vir die Voorsitter se afwesigheid. Elf vergaderings is in die jaar gehou. Daar is 'n groot verskuiwing na die goudvelde. 'n Gesamentlike vergadering by die Freddiesmyn in September 1951 was 'n sukses.

4. *Basoetoland*: 'n Brief van dr. J. de M. Vink is voorgelees.

Rede van die Uittredende President: Dr. J. N. W. Loubser het gepraat oor die rewolusie wat in 50 jaar plaasgevind het, 'n rewolusie op mediese, sosiologiese en ideologiese gebiede. In 1900 het medisyne net die dokter en sy pasiënt beteken. Vandag is die verhouding 'n sosiologiese een. In die ekonomiese stryd van vandag word ons beroepstatus soms vergeet. ('n Afskrif van die rede word in die Notuleboek ingeplak.)

Afsluiting van die rede is met toejuiging begroet. 'n Eendragtige opdrag van die vergadering was dat die Sekretaris die rede na die *Tydskrif* moet stuur vir publikasie.

Algemeen:

1. Die Sekretaris het briewe voorgelees: (a) Met betrekking tot bystandsverenigings. (b) Die vorming van 'n groep vir algemene praktisyns.

2. Dr. H. Dyke het voorgestel dat klein wapens ontwerp word om aan afgetrede Presidente van die tak te gee. Besluit is dat die takraad op die saak moet ingaan.

BY DIE KRUISPAD *

DR. J. N. W. LOUBSER

Dit is nou net 50 jaar gelede dat ek Suid-Afrika verlaat het om medisyne oorsee te gaan studeer, en dit mag miskien interessant en leersaam wees om terug te blik op die afgelope halfeeu en te sien wat alles in hierdie tydperk bereik en watter vorderings in die medisyne gemaak is. Ek voel ons professie het in meer as een opsig by die kruispad aangekom en dat vir die toekoms baie daarvan sal afhang watter uitdraaipad ons gaan kies.

Net soos op alle ander gebiede van die wetenskap en tegniek het ook ons vak gedurende die afgelope 50 jaar, sonder twyfel, meer vordering gemaak en veranderings ondergaan as gedurende die hele voorafgaande eeu, of selfs twee eeue. Dit geld egter ook op sosiale, ekonomiese en idiologiese gebiede, wat op hul beurt weer ons professionele benadering tot

* Rede van die Uittredende President, Tak Oranje-Vrystaat en Basoetoland, 23 Februarie 1952.

mediese vraagstukke, etiese begrippe en waardes byna rewolusionêr beïnvloed het en dit in die toekoms in nog sterkere mate sal doen.

Hierdie feite moet ons in die oë sien, daarmee rekenskap hou en ons daarby aanpas. Met die uitbreiding van ons mediese kennis en al die nuwe tegnies-ingewikkelde ondersoekings- en behandelingsmetodes is dit vir die individu heeltemal onmoontlik geword om vandag ook tot enige mate die hele mediese wetenskap te beheers. Dit het grootskaalse spesialisasie in feitlik alle afdelings van ons vak meegebring en noodsaaklik gemaak, maar gelyktydig ook ernstige omwentelinge in ons geledere veroorsaak.

Waar aan die begin van die eeu mediese behandeling nog uitsluitlik 'n saak tussen dokter en pasiënt was, word dit vandag hoe langer hoe meer 'n sosiale vraagstuk, waarin die Staat in toenemende mate 'n aandeel neem en in die toekoms nog meer gaan neem. In verskeie lande is daar reeds volledige staats-mediese dienste, waarin die dokter feitlik die rol van 'n staatsamptenaar speel.

In Suid-Afrika het ons alreeds vry hospitalisasie in die meeste van die provinsies; pogings om uitsluitlik voltydse dokters in hospitale aan te stel is al herhaalde kere in die verlede gemaak; die getal mediese hulpverenigings neem by die dag toe en daar is vingerwysings dat die dag miskien nie meer ver is nie wanneer die grootste deel van die bevolking in een groot staatsondersteunde mediese hulpvereniging opgeneem sal word nie.

Die voor- en nadele van so 'n skema is nie hier ter sprake nie, maar wel hoe ons professie ons by so 'n gebeurtenis gaan aanpas. Ons lewe vandag in 'n realistiese wêreld en soos in alle klasse van die samelewe is die ekonomiese stryd in ons geledere soms ook baie akuut en in hierdie stryd om die bestaan loop ons dikwels gevaar om ons beroepsstatus en professionele ideale uit die oog te verloor en te vergeet dat ons aan 'n edele professie en nie aan 'n werkersunie behoort nie.

Vergun my nou om slegs hier en daar enige van die vorderings wat die medisyne in die afgelope halfeeu gemaak het kortliks aan te stip. Alles het so gaandeweg geskied dat baie van ons miskien nie eens begryp het hoe rewolusionêr dit in menige opsig was nie. Behandelingsmetodes wat nog aan die begin van die eeu, ja selfs 25 jaar gelede, aan die orde van die dag was—ek wil hier net bv. die chirurgiese behandeling van tuberkuleuse gewrigte noem—is vandag heeltemal in onbruik; beskouings wat 'n 30 jaar gelede nog gehuldig was, is vandag ongeldig, verouder of vergeet; wat 'n 50 jaar gelede vir totaal onmoontlik gehou is, het nou werklikheid geword en baie vroeëre beskouings oor die ontstaan en behandeling van siektes is ons vandag geneig om as kinderagtige naïweteite te bestempel, sonder om te besef dat die volgende geslag dieselfde mag dink oor baie van wat ons nou as evangelie aanvaar.

Niks illustreer dit miskien beter nie as hoe vanselfsprekend die mediese professie, sowel as die publiek, die onlangse longreseksie op wyle koning George VI beskou het en die geweldige ophef wat daar in 1903 gemaak is toe koning Eduard VII 'n operasie vir 'n gewone akute appendicitis ondergaan het. En van akute appendicitis gepraat: dit laat my aan my studentejare terugdink hoedat daar selfs nog in 1906 'n hewige polemiek in al die mediese tydskrifte van Europa gewoed het oor die groot vraag of 'n operasie vir akute appendicitis in die akute stadium dan wel in die koorsvrye interval onderneem moes word, totdat von Bergmann—destyds as die gesaghebbendste chirurg van sy tyd beskou—kategories verklaar het: ‚Ek opereer 'n akute appendicitis wanneer ek dit in die hande kry,' en as bewys vir hierdie—vir ons vandag altans—byna vanselfsprekende opvatting, 50 agtereenvolgende geslaagde appendicektomie kon aanvoer.

Ofskoon Findlay, 'n algemene praktisyn op die eiland Kuba, reeds in 1881 met nadruk beweer het dat geelkoors deur 'n muskiet op die mens oorgedra word, is hy eenvoudig deur die deskundiges in Tropiese Siektes uitgelag, totdat Reid in 1905 deur 'n reeks baie deeglike toetse (wat selfs 'n paar menselewens gekos het) die bewys kon lewer dat Findlay reg en 'n huismuskiet, *Aëdes Egypti*, die sondebok is. Hierdie ontdekking het die bou—of liewer die voltooiing—van die Panamakanaal moontlik gemaak, al het dit ook al die oortuigingsvermoë van Gorgas gekos om president Theodore Roosevelt te beweeg om die nodige geld te bewillig om die kanaalstrook van muskiete te bevry.

Toe Schaudinn en Hoffmann in 1906 ontdek het dat die *Spirochaeta pallida* die verwekker van sifilis is, wou die mediese wêreld dit glad nie aanvaar nie en eers 'n paar jaar later, toe Schaudinn alreeds in sy graf was, is die juistheid van die ontdekking erken en Ehrlich in staat gestel om Salvarsan, beter bekend miskien as 606, die ‚wondermiddel teen sifilis' daar te stel. Sy verwagting dat 'n enkele inspuiting sifilis radikaal sou genees, is ongelukkig nie vervul nie; ook was 606 so giftig en het soveel lewens gekos dat dit 'n jaar of later deur Neosalvarsan (609) vervang is, wat onder 'n halfdosyn ander name nog steeds een van ons hoof wapens teen hierdie gésel van die mensdom bly.

Persoonlik het ek nog heelwat van die oorspronklike Salvarsan gebruik en kan alleen sê dat die resultate dramaties was. Nooit het ek meer as twee inspuitings nodig gevind om 'n ou verwaarloosde, destruktiewe rhinitis of vretende huidsifilied permanent te genees nie; en by ‚yaws' was die veranderings wat binne 12 uur na 'n inspuiting plaasgevind het byna ongelooflik. Die reaksies na 'n inspuiting het egter die dokter byna nog meer as die pasiënt aangetas.

Dit het my altyd ontstel hoe magteloos ons gestaan het teenoor gonoree—so 'n skynbaar eenvoudige, meestal plaaslik gelokaliseerde aandoening—wat egter onder omstandighede die grootste verwoesting kon aanrig. Hoe onbevredigend, langdurig en onsteties was die behandeling nie vroeër en hoe onseker die resultate nie. Wie sou 'n twintig jaar gelede ooit gewaag het om aan 'n pasiënt, en veral 'n vroulike pasiënt, wat aan gonoree gely het, 'n sertifikaat uit te reik, dat hy of sy genees en nie meer besmetlik is nie? Seker nie ek nie. Groot was dus my verrassing en verbasing toe ek omstreeks 1937 kennis gemaak het met Uleron (Bayer)—dit was 'n sulfapreparaat in tablet vorm wat per mond geneem word—en daarmee 'n akute, mikroskopies-gekontroleerde gonoree by 'n vroulike pasiënt binne 'n week kon genees. 'n Jaar of wat later het M & B 693 op die mark gekom en vandag beskik ons oor 'n reeks van sulfa-medikamente en antibiotika, van Penicillin tot Chloromycetin, en wie weet wat nog, as magtige wapens teen byna enige soort bakterië. Of die kieme ons nog op die duur sal uitoorlê en 'n immuniteit teen al hierdie spesifieke medikamente ontwikkel bly te sien, maar daar is nou al reeds aanduidings in hierdie rigting, net soos gerugte ook al gehoor word dat vlieë geleer het om hul ‚sokkies op te trek' as hulle met D.D.T. in aanraking kom.

Dit val swaar om te besef dat skaars veertig jaar gelede alle deskundiges nog geglo het dat beri-beri 'n aansteeklike siekte is en dat hierdie sienswyse vandag nog deur sommige dokters gehuldig word, of dat tot in die jaar 1925 dit nog sterk betwyfel was of pellagra nie 'n toksiese vergiftiging deur bederfde mielies was dan wel of dit deur 'n protozoon wat deur 'n klein vliegie op die mens oorgedra word, veroorsaak is. Dit is nogal interessant om die skynbaar oortuigende bewyse vir hierdie opvattings vandag te lees.

Die ontdekking van die vitamine en die belangrike rol wat hulle i.v.m. die gesondheid en stofwisseling van die liggaam speel, het 'n hele omwenteling in die medisyne teweeggebring en ons kennis van hierdie ‚spoorstowwe' brei nog daagliks uit; ons ken vandag alreeds die eienskappe en chemiese samestellings van meer as dertig van hulle. Ek wil my verstout om te voorspel dat verdere toekomstige ontdekkings op hierdie gebied en op die gebied van die hormone ons nog baie verrassings gaan besorg en nuwe lig werp op baie van die geheime van vatbaarheid vir aansteeklike siektes, rumatiek, chroniese arthritis, arteriosclerose, ens., en wie weet, miskien vind ons ook nog langs hierdie weg die sleutel tot die ontstaan van kankers.

Terloops, mag ek vra hoeveel van u het al daaraan gedink dat as dit nie was vir 'n vitamin, of liewer die gebrek aan 'n vitamin, ons nie vanjaar 'n van Riebeeck-fees sou vier nie?

Toe Banting en Best in 1922 insulin as 'n middel teen diabetes aan die medisyne geskenk het, het hulle seker nie kon droom nie dat dit 'n 20 jaar later as 'n doeltreffende middel vir die behandeling en selfs genesing van sekere vorms van kranksinnigheid, bv. dementia praecox gebruik sou word nie.

Gedurende die afgelope halfeeu is daarin geslaag om beskermende entstowwe teen byna alle aansteeklike siektes te ontdek (witseerkeel, pes, geelkoors, kinkhoes, tifus, cholera, ingewandskoors, om maar net 'n paar te noem) wat ons in staat stel om vatbaarheid vir hierdie siektes vir korter of langer periodes of selfs vir die res van die lewe te verhinder.

Na herhaalde mislukte pogings gedurende die afgelope 200 jaar is dit ons eindelik aan die begin van die eeu, deur die ontdekking van die bloedgroepe, geluk om bloedoortapping

veilig te maak, waardeur nie alleen jaarliks duisende lewens gered word nie, maar ons ook in staat gestel word om vandag operasies uit te voer wat ons nooit vroeër sou kon waag nie; en die toevallige ontdekking 'n jaar of tien gelede van die rhesus-faktoor in menslike bloed het ons nie alleen die verklaring gegee vir die sogenaamde hemolitiese siekte by pasgeborenes, menige doodgeboorte en habituele abortus nie, maar wat van byna nog meer belang is, dit het ook die stigma van kongenitale sifilis, wat ons in ons onkunde vroeër in al sulke gevalle as die oorsaak beskou het, eindelik verwyder.

As ons terugsien kan ons byna sê dat terwyl ons kollegas in die vorige eeu in die stryd teen siektes met voorlaaiers gewapen was, ons vandag oor agterlaaiers en masjiengewere beskik, en menigeen van ons mag miskien geneig voel om die bors 'n bietjie uit te stoot, maar voor ons dit doen laat ons eers die keersy van die medalje betrag en sien of ons daartoe geregtig is.

Ten spyte van ons vermeerde kennis en die legio van nuwe geneesmiddels waaroor ons beskik, vind ons dat ons hospitale oorvol is, selfs al het ons verlede jaar £4,000,000 op nuwe hospitaalgeboue gespandeer; die siektesyfers van die bevolking is maar nog steeds onrusbarend groot; die voorkoms- en sterftesyfer van tuberkulose styg nog elke jaar en geslagsiektes floreer in ons stede en op die platteland, veral onder nieblankes; geen week verloop nie of gevalle van pes, tifus, ingewandskoors, witseerkeel en selfs pokkies word deur die Departement van Gesondheid vermeld, en ons blanke kindersterftesyfer bly nog maar altyd in die buurt van 60 per 1,000. In Nu-Seeland is dit 30! Ek vrees ons bly nog maar altyd slegs geneeshere i.p.v. verhoeders van siekte en bewaarders van gesondheid, en van die predisponerende oorsake van siektes weet ons nog maar bloedweinig en bekommer ons skynbaar ook nie veel daaroor nie. Ja, ek kan u al hoor fluister ‚ondervoeding', en gee gewonne dat dit by sommige siektes, bv. tuberkulose, 'n belangrike rol mag speel, maar ek is nog lank nie oortuig dat dit die panasee vir die oplossing van al die probleme op hierdie gebied is nie. In hierdie opsig moet ek altyd weer aan die 1918 griepepidemie terugdink en hoedat die kinders en ou mense byna 'n absolute immuniteit teen die siekte getoon het, terwyl die gesonde, goedgevoede deel van die bevolking in die fleur van die lewe daardeur afgemaai is. En wat van kinderverlamming? Is dit die ondervoede jeug van die stedelike agterbuurte of die kinders van die gegoede, goedgevoede deel van die bevolking wat die meeste slagoffers lewer? Nee, ek vrees daar is nog 'n faktoor X, waarvan ons vandag nog maar baie min weet, en miskien sal die ontdekking hiervan vir ons eendag nog net so 'n openbaring wees as wat die rhesus-faktoor was.

'n Ander saak wat selfs vandag nog veels te min die aandag van dokters geniet is die rol wat psigosomatiese en somatopsigiese disorganisasie in die ontstaan en veral in die bestendiging van baie siektes speel; hoeveel chroniese invalides is nie die slagoffers van die onoordeelkundige gebruik, 'n mens kan byna sê misbruik, van die sfigometer of die elektrokardiograaf deur die sigologies-ongeskoolde huisdokter of spesialis-internis nie?

Nog 'n probleem van die tyd is die kwessie van spesialisasie in ons beroep. Soos in elke ander wetenskap, bestaan daar ook in die medisyne die nouste wisselwerking tussen spesialisering en vooruitgang; die een is byna nie sonder die ander denkbaar nie, ja 'n mens kan sê dat die twee terme vandag sinonieme geword het. Met die huidige ontwikkeling in ons vak kan geen enkeling meer die hele gebied ook maar enigsins volledig beheers nie en dit is alleen wanneer sekere lede van die professie hul uitsluitlik by een of ander vertakking daarvan bepaal dat die voordele van al die nuwe ingewikkelde ondersoekings- en behandelingsmetodes die pasiënt behoorlik ten goede kom.

Ongelukkig is die posisie in ons professie ietwat uniek in so ver dat die spesialis en die algemene praktisyn hoe langer hoe meer met mekaar begin kompeteer. So iets het ons bv. glad nie in die wetsprofessie nie. Van die spesialis word geverg dat hy sy spesialiteit ten volle moet beheers en hom daarby moet bepaal; hy is ook egter geregtig om aansienlik meer as die algemene praktisyn vir sy dienste te vra. Van die algemene praktisyn weer word verwag dat hy 'n deeglike algemene kennis van die hele mediese wetenskap moet besit en hy het die reg om enige mediese handeling, waartoe homself deur ondervinding of opleiding bevoeg geag, te onderneem. Dit word egter ook van hom geverg dat hy die grense van sy kennis nie sal oortree en handelings onderneem wat bo sy vuurmaakplek is nie, maar sulke gevalle na 'n bevoegde spesialis sal verwys. Juis hier lê die knoop; en botsings tussen etiese begrippe en ekonomiese eiebelang vind helaas maar al te dikwels plaas en ek vrees dat laasgenoemde nog veels te veel die slagveld behou—tot nadeel van die pasiënt.

Dit is byna vanselfsprekend dat in 'n jong land soos Suid-Afrika spesialisasie veel later as in die ouere lande van Europa ontwikkel het. Aan die begin van die eeu was daar feitlik maar net een bekende en erkende spesialis, nl. die oogarts; hierna het die chirurg gevolg en gaandeweg en met steeds versnellende tempo het meer as 700 praktiserende spesialiste in omtrent 20 spesialiteite op die toneel verskyn, d.w.s. een spesialis vir elke agt algemene praktisyns, en gevolglik is daar vandag skerp kompetisie tussen hierdie twee groepe in die professie. Aan die een kant laak die spesialis dit dat behandelings, en veral operatiewe behandelings, wat onder sy spesialiteit sorteer, deur die algemene praktisyn onderneem word. Aan die ander kant het dit vandag gebruiklik geword dat die spesialis nie net pasiënte wat deur die huisdokter na hom verwys is, ondersoek en behandel nie, maar dat die publiek in toenemende mate direk na hom vir raad en behandeling gaan, en dit ook ontvang. As gevolg hiervan voel die algemene praktisyn dat sy professionele status ondermyn en sy bestaan bedreig word en dat as dinge so aanhou hy eindelik net 'n soort van informasieburo sal word om pasiënte te sorteer en na die betrokke spesialis te verwys.

Hierdie vraagstuk van spesialis versus algemene praktisyn het gedurende die afgelope paar jare by ons in Suid-Afrika sowel as in verskeie ander lande die ernstige aandag van die professie geniet, maar ek vrees dat tot datum nog geen bevredigende oplossing gevind is nie. Miskien lê dit in die rigting van 'n persentasiebeperking van spesialiste in verhouding tot algemene praktisyns en die bepaling dat spesialiste net as konsultante mag optree soos bv. advokate in die wetsprofessie doen. Een ding mag nie uit die oog verloor word nie en dit is dat sonder die algemene praktisyn geen doeltreffende mediese diens denkbaar is nie en dat as die algemene praktisyn eenmaal van die toneel verdwyn het dit feitlik onmoontlik sal wees om hom ooit weer in die lewe terug te roep.

Toe ek 'n 25 jaar gelede in Europa was, het dit my getref hoe baie mense gekla het dat 'n goeie huisdokter onverkrygbaar was omdat die spesialiste sy bestaan onmoontlik gemaak het; en enige jare gelede het 'n hooggeplaaste Suid-Afrikaanse amptenaar in Italië my die volgende ondervinding meegedeel: Een nag in 'n hotel in Milaan word sy vierjarige seuntjie siek met 'n seer keel. Bevrees dat dit witseerkeel kon wees het hy die hotelbaas versoek om 'n dokter te bel. Na 'n rukkie verskyn die dokter, maar toe hy merk dat dit 'n kind is, gee hy sy spyt te kenne dat hy die pasiëntjie nie mag ondersoek nie aangesien hy geen kinderspesialis is nie. Hy word betaal en gaan. 'n Kinderspesialis word nou ingeroep, maar toe hy verneem dat hy 'n seer keel het deel hy die ouers mee dat, hoewel 'n kinderarts, hy nie 'n keelspesialis is nie en gevolglik ook nie die kind mag ondersoek nie. Ook hy word betaal en vertrek. Eindelik verskyn 'n keelspesialis op die toneel en stel vas dat die kind 'n gewone tonsillitis het. Wat 'n ‚reductio ad absurdum'. En as ons nie oppas nie sal ons een van die dae soortgelyke gevalle ook in Suid-Afrika beleef.

Met die hedendaagse verbeterde maar tewens ook ingewikkelder en gespesialiseerder ondersoekings- en behandelingsmetodes het die koste van mediese behandeling aansienlik gestyg en dit ly geen twyfel nie dat die middelstand dit dikwels uiters moeilik vind om dit te bekostig. Ook vat die idee hoe langer hoe meer pos dat dit die plig van die Staat is om vir die gesondheid van die bevolking te sorg. Baie lande het reeds vandag staatsmediese dienste en in Suid-Afrika het die Gluckman-kommissie enige jare gelede 'n volledige staatsmediese diens aanbeveel ofskoon dit intussen nie aanvaar is nie. Alles dui egter vandag weer daarop dat die tyd nie meer ver is nie dat 'n staatsmediese diens in een of ander vorm ingevoer sal word. Dit maak nie saak wat ons persoonlik oor so 'n skema mag voel nie, die gety van die tyd sal ons nie kan weerstaan nie. Wat ons egter kan en behoort te doen, is om intussen 'n deeglike studie van soortgelyke dienste in ander lande te maak sodat as die tyd aanbreek ons met gesag kan praat en leiding kan gee om 'n skema wat in die beste belange van beide die bevolking en die professie sal wees, te verkry.

As u met my mag saamstem dat ons in meer as een opsig vandag by die kruispad aangekom het, en my sou vra watter rigting sou gekies moet word, dan sou ek dit kortliks so wou stel:

1. Die invloed van sosiale faktore op gesondheid moet in die toekoms ons volste aandag geniet.

2. In belang van die professie sowel as van die publiek is dit dringend noodsaaklik dat 'n bevredigende, praktiese oplossing wat die status en bestaan van die algemene praktisyn vrywaar, gevind word.

3. Terwyl ons kollegas in die vorige eeu hoofsaaklik net by magte was om *siekteverskynsels* te behandel, het ons in die afgelope 50 jaar die kennis opgedoen en die middels verkry om *siektes* te behandel; in die vervolg moet die *verhoeding van siektes* en die beskerming van gesondheid ons ideaal en strewe wees.

PASSING EVENTS

The British Medical Association has advised the Medical Secretary that it has been found necessary to increase the subscription rate payable by *bona fide* medical students from 10s. 6d. per annum to £1 1s. per annum. This change came into effect on 23 January 1952 and will affect all new student subscribers and those who renew their subscriptions after that date.

Medical Research Scholarship

Applications are invited for the Medical Research Scholarship awarded annually by the Grocers' Company. This is of the value of £450 for the first year of tenure and of £650 in the second, if the holder be reappointed; a further allowance of up to £100 each year is made for research expenses.

Applicants must be British subjects and not over 35 years of age. Preference is given to those not holding other remunerative employment, and thus able to devote the whole of their time to the proposed research.

The tenure of the Scholarship begins annually on 1 September.

Applications must reach London by 1 June 1952.

Further particulars, conditions of tenure, etc., are available from the Assistant Registrar, University of the Witwatersrand.

REVIEWS OF BOOKS

Vaginal Cytology

Gestation et Cytologie Vaginale. By J. Paul Pundel, Fred van Meensel and Z. Jaworski. (Pp. 209. 2,250 Fr.) Paris: Masson et Cie.

Interest in vaginal cytology throughout the menstrual cycle and during pregnancy continues to be reflected in excellent communications from the American as well as the Continental literature.

The present monograph is a contribution to the normal and abnormal changes which occur. There is a wealth of illustration of the histology of the vagina, often side by side with the corresponding picture of the endometrium. There are also very beautiful colour plates illustrating smears.

The monograph is an invaluable addition to physiology as well as pathology.

South African Periodicals

Handlist of South African Periodicals Current in December 1951. Compiled by C. Daphne Saul, F.S.A.L.A. Grey Bibliographies No. 5. (Pp. 54. 3s. 6d.) Cape Town: South African Public Library. 1951.

The publication of this list of the South African periodicals fulfils a particularly valuable function because so many Journals have either ceased publication or changed their editors, and so many more have appeared for the first time since the handbook was last published in 1945.

As Mr. Varley points out in his foreword, the present list is virtually a new publication. Periodicals entirely local in appeal such as newspapers, school and University magazines, parish and local church magazines, etc., have been omitted.

This handbook should prove extremely useful to those faced with South African bibliographical problems.

Atlas of Anatomy

An Atlas of Anatomy. By J. C. Boileau Grant, *M.C.*, M.B., Ch.B., F.R.C.S. (Edin.). (Pp. 503 + xiv, with 637 figures, some in colour. Third Edition. 91s. 6d.) London: Baillière, Tindall & Cox. 1951.

Contents: Illustrations. 1. The Upper Limb. 2. The Abdomen. 3. The Perineum and Pelvis. 4. The Lower Limb. 5. The Vertebrae and the Vertebral Column. 6. The Thorax. 7. The Head and Neck. 8. The Cranial Nerves and the Dermatomes.

It is a great pleasure to welcome the third edition of this useful and standard work, which is improved by over 70 new illustrations, with replacement of and addition of colour to some of the older ones.

The reproduction of the illustrations, needless to say, maintains the superb standard of the earlier edition.

Questions Answered

Any Questions? A Selection of Questions and Answers Published in the British Medical Journal. First Series. (Pp. 240. 7s. 6d.) London: British Medical Association. 1951.

Contents: 1. Allergy. 2. Anaesthesia. 3. Blood Disorders. 4. Cancer. 5. Cardiovascular System. 6. Dermatology. 7. Ear, Nose and Throat. 8. Endocrinology. 9. Fevers. 10. Forensic Medicine and Toxicology. 11. Gastro-Intestinal Diseases. 12. General Medicine. 13. Heredity and Disease. 14. Immunization. 15. Neurology. 16. Nutrition. 17. Ophthalmology. 18. Orthopaedics. 19. Paediatrics. 20. Tuberculosis. 21. Urinary Disorders. 22. Venereal Disease. 23. Worms. 24. Final Miscellany.

A valuable feature in the *British Medical Journal* is the series of *Questions* and *Answers* published from time to time. A selection of these has now been gathered together and published as a first series. They have been carefully classified (as the Table of Contents indicates) and they cover such practical features of medicine so authoritatively, widely and well that the volume is bound to be received with great pleasure.

The book is an excellent companion to the undergraduate medical student and will grace the library shelf of every practising doctor.

Hormones

Hormones: A Survey of Their Properties and Uses. Published by direction of the Council of the Pharmaceutical Society of Great Britain. (Pp. 220 with 34 illustrations and 14 tables. 35s.) London: The Pharmaceutical Press. 1951.

Contents: 1. Introduction. 2. History. 3. Physiology. 4. Chemistry of the Non-Steroid Hormones. 5. Chemistry of the Steroid Hormones. 6. Standardization. 7. Action and Uses. 8. Pharmacy. 9. Commercial Preparations. 10. Bibliography. 11. Index.

This excellent monograph has been published by the Pharmaceutical Press by direction of the Council of the Pharmaceutical Society of Great Britain. It is clearly written, intelligently illustrated with structural formulae and pertinent graphs. It is invaluable for those who seek a simple and lucid introduction to the modern complexities of hormones.

A final chapter includes a list of commercial productions and the very good bibliography is an excellent guide to those who wish to pursue their studies further. The chapter on *Standardization* gives up-to-date information about international standards and serves as a very good introduction to the study of biological methods of assay.

This book can be recommended strongly.

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(992) South-Eastern Cape hospital town. Premium required £1,500 which includes drugs, furniture and instruments worth approximately £1,350. Flat plus surgery to let at £6 p.m.

(993) Noord-Kaapland. Dorp met privaat verpleeginrigting. Gemiddeld £200 p.m. kontant ontvangste. Koopprys van £5,500 sluit in huis en 2 aparte geboue, meubels, instrumente, medisyne en praktyk.

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Three years' service after qualification: £600 per annum, plus free quarters or an allowance in lieu thereof.

Four years' service after qualification: £700 per annum, plus free quarters or an allowance in lieu thereof.

Five or more years' service after qualification: £800 per annum, plus free quarters or an allowance in lieu thereof.

In addition to the foregoing salary, a temporary cost-of-living allowance is also payable.

Applications, giving full details of experience and qualifications, should be addressed to the Director of Provincial Medical and Health Services, P.O. Box 20, Pietermaritzburg, to reach him not later than 30 April 1952.

AD.6938

S.A. Medical Journal
S.A. Tydskrif vir Geneeskunde

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Author's reprints of papers can be obtained at cost. Order blanks will be forwarded to authors when page proofs are ready.

Provincial Administration of the Cape of Good Hope/University of Cape Town:

JOINT MEDICAL STAFF

1. Applications are invited for appointment to a post of Medical Practitioner, Grade F (Department of Medicine), with salary at the rate of £1,800 per annum (fixed) on the Joint Staff for the Groote Schuur Hospital and other teaching hospitals in the Cape Peninsula.

2. In addition to the salary indicated cost-of-living allowance at rates prescribed from time to time by the Administrator is payable to whole-time officials and employees. Present rate: married persons, £256 per annum, and single persons, £80 per annum.)

3. The conditions of service are prescribed in terms of the Hospital Board Service Ordinance No. 19 of 1941, as amended, and the regulations framed thereunder.

4. (a) The Joint Medical Staff will be required to serve jointly the Provincial Administration of the Cape of Good Hope and the University of Cape Town.

(b) A session shall be four hours per week, not necessarily continuous, of clinical and/or teaching work.

5 (a) Candidates must state whether they wish to be considered for—

i. appointment in a whole-time capacity; or

ii. appointment in a part-time capacity; or

iii. appointment either in a whole-time capacity or in a part-time capacity.

(b) Should they wish to be considered for appointment in a part-time capacity, the maximum number of sessions which they would on appointment be prepared to give, indicating preference for days and times, should also be stated.

6. The successful candidates will be required to submit satisfactory birth and health certificates.

7. Applications must be made on the prescribed form Staff 23 which is obtainable from the Director of Hospital Services, P.O. Box 2060, Provincial Building, Wale Street, Cape Town, or from the Branch Representative of the Hospitals Department at Cape Town (P.O. Box 1487), East London (P.O. Box 13), Port Elizabeth (P.O. Box 80), Kimberley (P.O. Box 618), and Umtata (P.O. Box 202), or from the Medical Superintendent of any Provincial Hospital or Secretary of any School Board in the Cape Province.

8. The completed application forms must be addressed to the Director of Hospital Services, P.O. Box 2060, Cape Town, and must reach him not later than 30 April 1952. Candidates must state the earliest date on which they can assume duty.

Y 248527

Provinsiale Administrasie van die kaap die Goeie Hoop/Universiteit van Kaapstad:

GESAMENTLIKE MEDIESE PERSONEEL

1. Aansoeke word ingewag vir aanstelling tot die pos van Geneesheer, Graad F (Departement van Medisyne), met salaris teen £1,800 per jaar (vasgestel) op die Gesamentlike Mediese Personeel by die Groote Schuur-hospitaal en ander opleidingshospitale in die Skiereiland.

2. Benewens die salarisskaal soos aangedui, is 'n lewenskostetoelae teen tariewe wat van tyd tot tyd deur die Administrateur vasgestel word, betaalbaar aan voltydse beamptes en werknemers. Teenswoordige tarief: getroude persone, £256 per jaar, en enkel persone, £80 per jaar.)

3. Die diensvoorwaardes word voorgeskryf ingevolge die Ordonnansie op Hospitaalraadsdiens nr. 19 van 1941, soos gewysig, en die regulasies wat daarkragtens opgestel is.

4. (a) Van die Gesamentlike Mediese Personeel sal vereis word om die Provinsiale Administrasie van die Kaap die Goeie Hoop en die Universiteit van Kaapstad gesamentlik te dien.

(b) 'n Sessie is vier uur per week in verband met kliniese en/of opleidingswerk maar is nie noodwendig onafgebroke nie.

5. (a) Kandidate moet meld of hulle in aanmerking geneem wil word vir—

i. aanstelling in 'n voltydse hoedanigheid; of

ii. aanstelling in 'n deeltydse hoedanigheid; of

iii. aanstelling òf in 'n voltydse òf in 'n deeltydse hoedanigheid.

(b) As hulle in aanmerking geneem wil word vir aanstelling in 'n deeltydse hoedanigheid, die maksimum getal sessies wat hulle by aanstelling gewillig sal wees om by te woon, asook die dae en tye wat hulle verkies.

6. Die suksesvolle kandidate moet bevredigende geboorte- en gesondheidsertifikate indien.

7. Aansoek moet gedoen word op die voorgeskrewe vorm Staf 23 wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Provinsiale Gebou, Waalstraat, Kaapstad, of by die Takverteenwoordiger van die Hospitaaldepartement te Kaapstad (Posbus 1487), Oos-Londen (Posbus 13), Port Elizabeth (Posbus 80), Kimberley (Posbus 618), en Umtata (Posbus 202), of by die Mediese Superintendent van enige Provinsiale Hospitaal of die Sekretaris van enige Skoolraad in die Kaapprovinsie.

8. Die voltooide aansoekvorms moet gerig word aan die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, en moet hom nie later as 30 April 1952 bereik nie. Kandidate moet die vroegste datum meld waarop hulle diens kan aanvaar.

Y 248527

Provincial Administration of the Cape of Good Hope

HOSPITALS DEPARTMENT

1. Applications are invited from medical graduates for appointment to the posts of Junior Resident Medical Officer (Intern) at the undermentioned institutions:—
Groote Schuur Hospital.
Somerset Hospital (General and Maternity Section).
Woodstock Hospital.
Rondebosch and Mowbray Hospitals.
Victoria Hospital, Wynberg.
False Bay Hospital, Simonstown.
Peninsula Maternity Hospital.
Mowbray Maternity Hospital.
2. The salaries attaching to the posts are £240 per annum plus board, quarters and laundering.
3. In addition to the salaries and allowances stated above a temporary non-pensionable cost-of-living allowance is payable at the rates and on conditions that may be prescribed by the Administrator.
4. Applicants applying for more than one post should submit separate applications and copies of testimonials for each post applied for.
5. The successful applicants will be required to enter into contracts with the Provincial Administration with effect from 16 July 1952, and must be registered with the South African Medical Council before they will be allowed to assume duty.
6. No canvassing is permitted.
7. The appointments are governed by Ordinance No. 19 of 1941, as amended from time to time and by the regulations framed thereunder.
8. Applications must be made on the prescribed form Staff 23 which is obtainable from the Director of Hospital Services, P.O. Box 2060, Provincial Building, Wale Street, Cape Town, or from the Branch Representative of the Hospitals Department at Cape Town (P.O. Box 1487), East London (P.O. Box 13), Port Elizabeth (P.O. Box 80), Kimberley (P.O. Box 618), and Umtata (P.O. Box 202), or from the Medical Superintendent of any Provincial Hospital or Secretary of any School Board in the Cape Province.
9. Applications containing particulars of experience, qualifications, etc., should be forwarded to reach the Medical Superintendent of the institutions concerned not later than noon on 29 April 1952.

Y 248528

Provinsiale Administrasie van die Kaap die Goeie Hoop

HOSPITAALDEPARTEMENT

1. Aansoeke word ingewag van mediese gegradueerdes vir aanstelling in die betrekkings van Junior Inwonende Mediese Beampte (Intern) aan die ondergemelde inrigtings:—
Groote Schuur-hospitaal.
Somerset-hospitaal (Algemene en Kraamafdeling).
Woodstock-hospitaal.
Rondebosch en Mowbray-hospitaal.
Victoria-hospitaal, Wynberg.
Valsbaai-hospitaal, Simonstad.
Skiereilandse Kraaminrigting.
Mowbray-kraaminrigting.
2. Die salarisse verbonde aan die poste is £240 per jaar plus losies, inwoning en wasgoed.
3. Benewens die bovermelde salarisse en toelaes is daar 'n tydelike nie-pensioengewende duurtetoeslag betaalbaar volgens die skaal en voorwaardes wat deur die Administrateur van tyd tot tyd voorgeskryf word.
4. Applikante wat om meer as een betrekking aansoek doen, moet afsonderlik aansoek en afskrifte van getuigskrifte voorlê vir elke betrekking waarom aansoek gedoen word.
5. Van die geslaagde applikante word vereis om 'n kontrak met die Provinsiale Administrasie met ingang van 16 Julie 1952 aan te gaan en hulle moet geregistreer wees by die Suid-Afrikaanse Mediese Raad voordat hulle toegelaat sal word om diens te aanvaar.
6. Geen stemmewerwing word toegelaat nie.
7. Die aanstellings is ooreenkomstig Ordonnansie nr. 19 van 1941 soos van tyd tot tyd gewysig en die regulasies wat daarkragtens opgestel is.
8. Aansoek moet gedoen word op die voorgeskrewe vorm Staf 23 wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Provinsiale Gebou, Waalstraat, Kaapstad, of by die Takverteenwoordiger van die Hospitaaldepartement te Kaapstad (Posbus 1487), Oos-Londen (Posbus 13), Port Elizabeth (Posbus 80), Kimberley (Posbus 618), en Umtata (Posbus 202), of by die Mediese Superintendent van enige Provinsiale Hospitaal of die Sekretaris van enige Skoolraad in die Kaapprovinsie.
9. Aansoeke met volledige besonderhede van ondervinding, kwalifikasies, ens., moet gerig word aan die Mediese Superintendent van die betrokke inrigting en moet hom nie later as 12-uur middag op 29 April 1952 bereik nie.

Y 248528

City of Johannesburg

VACANCY

Applications are invited from Europeans for the following vacant position in the City Health Department:—

Temporary Medical Officer (Native Townships Clinics): Salary £996 per annum fixed, plus cost-of-living allowance (at present £25 9s. 5d. per month), plus locomotion allowance.

Applicants must be medical practitioners registered to practise in the Union.

Details of conditions of service may be had on application from the Medical Officer of Health, P.O. Box 1477, Johannesburg.

Personal canvassing for appointment in the gift of the Council is strictly prohibited. Proof thereof shall disqualify a candidate for appointment.

Applications in the candidate's own handwriting on special forms to be obtained from the Central Staff Office, Room 223, Municipal Offices, must be placed in the box in Room 223, Municipal Offices, or posted so as to reach the undersigned not later than 12 noon on 26 April 1952.

Brian Porter
Town Clerk
380/1029

The Divisional Council of the Cape

VACANCY FOR HOUSE PHYSICIAN

DR. A. J. STALS MEMORIAL SANATORIUM

Applications are invited from suitably qualified persons for the undermentioned vacancy at the Dr. A. J. Stals Memorial Sanatorium, Retreat.

House Physician: Fixed salary of £360 per annum plus cost-of-living allowance, less £96 per annum for quarters and rations. Appointment for six months' duration, not regarded as internship. Married quarters are available.

The services of the successful applicant will be required as soon as possible after the closing date.

Applications should be addressed to reach the undersigned not later than noon on 5 May 1952.

Canvassing of Councillors or officials will be a disqualification.

G. O. Owen
Secretary

6 Dorp Street
Cape Town
7 April 1952

Transvaalse Provinsiale Administrasie

VAKATURES BY PUBLIEKE HOSPITALE

Aansoeke word ingewag van kandidate met geskikte kwalifikasies vir die onderstaande poste by Publieke Hospitale in die Transvaal.

Aansoeke moet gerig word aan die Geneeskundige Superintendent of Verantwoordelike Geneesheer van die betrokke Hospitaal en moet volle besonderhede bevat aangaande die ouderdom, professionele, akademiese en taalkwalifikasies, ondervinding en huwelikstaat van die applikant en moet voorts 'n aanduiding bevat van die vroegste datum waarop diens aanvaar kan word.

Hospitaal	*Vakature*	*Salaris*	*Aanmerkings*
Barberton	Verantwoordelike Geneesheer (1)	£1,000 × 50 —1,200	Moet 'n geregistreerde mediese praktisyn wees. Plus £180 per jaar huistoelae. Getroud plus (*a*) hieronder. Ongetroud plus (*b*) hieronder.
Bethal	Verantwoordelike Geneesheer (1)	£1,000 × 50 —1,200	Moet 'n geregistreerde mediese praktisyn wees. Plus £180 p.j. huistoelae. Getroud plus (*a*) hieronder. Ongetroud plus (*b*) hieronder.
Edenvale, PK. Raedene	Kliniese Patoloog (1)	£1,800 p.j.	Die pos sal aan die Edenvale-hospitaal verbonde wees en die bekleër van pos moet beskikbaar wees vir diens by enige hospitaal soos deur die Direkteur van Publieke Hospitale neergelê. Getroud plus (*a*) hieronder. Ongetroud plus (*b*) hieronder.
Klerksdorp	Junior Radioloog (Rondreisende) (1)	£1,200 × 50 —1,500	Om diens te doen by die Klerksdorpse, Potchefstroomse en Wolmaransstadse hospitale. Getroud plus (*a*) hieronder. Ongetroud plus (*b*) hieronder.
Potgietersrust	Deeltydse Algemene Praktisyn (1)	£510 p.j.	Om hospitaal daagliks te besoek. Om 12 uur aan hospitaal per week te bestee.
Pretoria	Deeltydse Senior Kinderarts (1)	£615 p.j.	Moet 'n geregistreerde mediese praktisyn wees. Drie sessies per week.
Pretoria	Ongevalle Beamptes (2)	£620—780 —820—860	Moet geregistreerde mediese praktisyns wees. Getroud plus (*a*) en (*c*) hieronder. Ongetroud plus (*b*) hieronder.

(*a*) £256 per jaar lewenskostetoelae.
(*b*) £80 per jaar lewenskostetoelae.
(*c*) Tydelike toelae.

Van persone wat aangestel word, sal verwag word om bevredigende sertifikate in te dien, asook om hulle te onderwerp aan 'n geneeskundige ondersoek by die betrokke hospitaal.

Aansoekvorms is verkrygbaar van die Provinsiale Sekretaris, Departement van Hospitaaldienste, Posbus 383, Pretoria.

Benewens jaarlikse salaris ontvang voltydse werknemers op die oomblik lewenskostetoelae, spoorwegkonsessie en word verlof toegestaan ooreenkomstig die hospitaal verlofregulasies.

Die sluitingsdatum van aansoeke vir die poste is 28 April 1952.

34651

Public Service Commission

VACANCIES IN THE PUBLIC SERVICE

1. The attention of medical practitioners, registered with the South African Medical and Dental Council, is drawn to an advertisement appearing in the *Government* and *Provincial Gazettes* of this week, inviting applications for the undermentioned posts.

Post	*Department/ Administration*	*Salary Scale* £
Medical Inspector of Schools	Cape Provincial Administration	950 × 50—1,300
Medical Inspector of Schools	South West Africa Administration	950 × 50—1,300
Medical Officer	Health (King George V Hospital, Durban and Nelspoort Sanatorium)	900 × 50—1,150
Medical Officer	Health (Mental Hospital Service)	900 × 50—1,150

2. In addition to salary a cost-of-living allowance at the rate of £256 per annum (married) and £80 per annum (single) is payable at present.

3. It is emphasized that full and detailed particulars of qualifications and previous experience (including military service) must be furnished but original certificates and testimonials should not be submitted. Application forms Z.83 and P.S.C. 8 (*a*) are obtainable from the Secretary, Public Service Commission, Pretoria, to whom filled-in forms must be addressed.

4. The closing date for the receipt of applications is 10 May 1952.

34642

City of Johannesburg

CITY HEALTH DEPARTMENT

VACANCY : PART-TIME RADIOLOGIST

Applications are invited for the position of part-time Radiologist. Applicants for the position must be registered with S.A. Medical and Dental Council as specialists in Radiology.

The duties will involve approximately 12 hours per week at the Council's non-European hospital at Waterval.

The remuneration will be £684 per annum fixed.

Details of conditions of service will be supplied on application to the Medical Officer of Health, Room 220, 18 Hoek Street (or P.O. Box 1477), Johannesburg.

Personal canvassing for appointments in the gift of the Council is strictly prohibited. Proof thereof shall disqualify a candidate for appointment.

Applications on special forms to be obtained from the Central Staff Office, Room 223, Municipal Offices, must be forwarded to Room 223, Municipal Offices, not later than 30 April 1952.

Brian Porter
Town Clerk
331/1008

Dorpsbestuur van Loeriesfontein

DEELTYDSE GENEESKUNDIGE GESONDHEIDSBEAMPTE

Aansoeke word deur die ondergetekende ingewag tot 21 April 1952 om die vakante betrekking as deeltydse Geneeskundige Gesondheidsbeampte vir die Dorpsbestuur van Loeriesfontein. Salaris £5 per maand. Meld volle besonderhede aangaande kwalifikasies, ondervinding en tweetaligheid. Verdere besonderhede van pligte en voorwaardes van aanstelling kan by die ondergetekende verkry word.

C. Koegelenberg
Sekretaris

Kantoor van die Dorpsbestuur
Loeriesfontein
26 Maart 1952

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When chronic worry retards recovery . . .

Chronic worry frequently stands stubbornly in the way of a patient's recovery from illness and forms a troublesome part of the total clinical picture. 'Drinamyl'—a balanced combination of 'Dexedrine' and amylobarbitone—will help you to combat this problem since it modifies extremes of behaviour and brings about an improved outlook, thus enabling the patient to cope more readily with day to day difficulties. During this period the relief of symptoms is a stimulus to recovery.

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